THE PLANT DISEASE REPORTER

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so.

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ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 12-double-spaced typed pages, including tables, graphs, and photographs. Because of reproduction costs photographs should be kept to a minimum. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

Manuscripts for and correspondence about this publication should be sent to:

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Mycology and Plant Disease Reporting Section
Crops Protection Research Branch
Plant Industry Station
Beltsville, Maryland

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CONTENTS

ELSINOË DISEASE OF SUGARCANE IN FLORIDA

Edwin H. Todd[1]

A species of Elsinoë[2] on sugarcane, Saccharum officinarum, was brought to the attention of the writer in June 1959. Since that time the disease it causes has been found in commercial fields of sugarcane varieties F. 31-436 and Cl. 41-223 at Canal Point, Florida. The identity of the causal fungus from Florida was confirmed by Anna E. Jenkins, an authority on species of Elsinoë.

Elsinoë infections on sugarcane are first recognized by the presence of minute purple lesions usually on the upper side of leaf and midrib tissues. A stereoscopic microscope reveals that these lesions later coalesce and become erumpent with irregular, ovoid to elliptical white spored fruiting bodies, usually measuring less than 1/4 to 1 1/2 mm; the fruiting bodies have thick, dark-brown margins. To the naked eye the fructifications appear as a white rash (Fig. 1). The whole or only portions of the leaf may be infected.

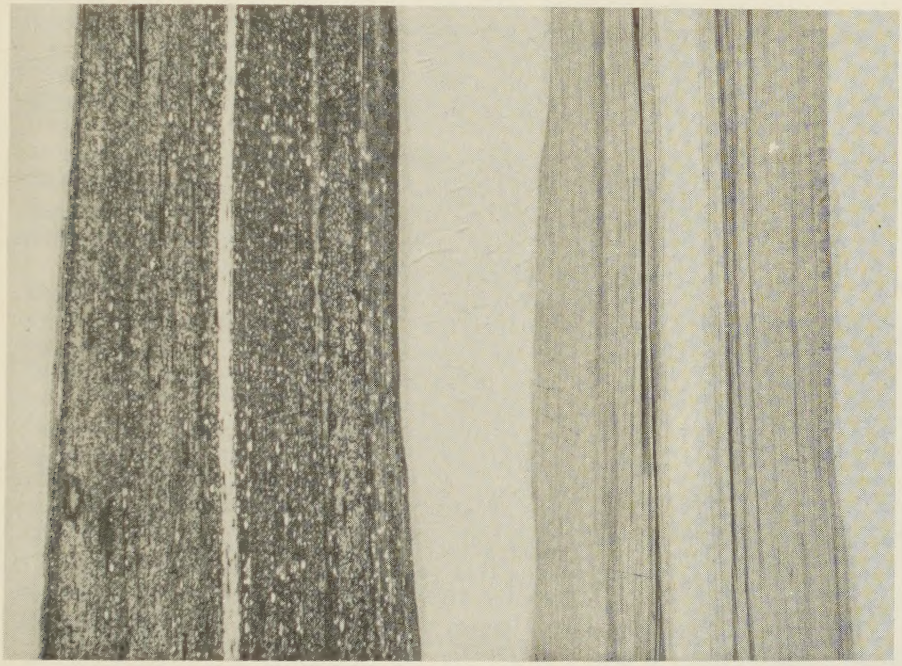

FIGURE 1. Left -- leaf of sugarcane variety F. 31-436 infected with Elsinoë sp. Right -- healthy leaf of the same variety.

The disease was fairly prevalent in a field of mature F. 31-436 sugarcane at Canal Point. On this variety, however, the fungus was found on only the lower leaves of mature stalks, on leaves that had been damaged by wind or other causes, or on leaves infected and apparently weakened by other fungi, such as species of Helminthosporium. Occasionally infections were found on the under side of leaves and on leaf sheaths. The fungus was not found on the stalks. Thus far, the writer has not observed the disease on any other varieties in commercial fields or in the World Collection of sugarcane at Canal Point, including the parental varieties of F. 31-436 and Cl. 41-223. However, the same species of Elsinoë probably will be found later on other varieties of the Collection.

Dr. Jenkins recently discovered Elsinoë infections on specimens of sugarcane from various parts of the world in the herbarium at Beltsville, Maryland. Some of these specimens were collected at Canal Point by B. A. Bourne in 1929. There is reason to assume, then, that the present disease of sugarcane at Canal Point has existed, though hitherto unrecognized, for many years. These limited observations and available data suggest that Elsinoë of sugarcane is at best a weak parasite of minor economic importance in the vicinity of Canal Point, Florida.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

[1] Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[2] A description of the fungus is being prepared by S. C. Arruda, who first discovered it on sugarcane in Brazil. Dr. Arruda's finding is the first report of Elsinoë sp. on a species of Gramineae.

TRANSMISSION OF THE ASTER YELLOWS VIRUS TO BARLEY[1]

E. E. Banttari and M. B. Moore[2]

The authors reported previously that, in addition to the transmission of the oats blue dwarf virus to oats and to barley, a second disease of barley was found in which plants were severely dwarfed, internodes failed to elongate, and the leaves rolled tightly backward[3]. It appeared that this disease was caused by a leafhopper-transmitted virus acting either alone or in combination with the oat blue dwarf virus. The oat blue dwarf virus alone did not cause severe dwarfing or backward leaf roll in barley.

It has now been demonstrated that this "leaf roll dwarf" of barley is caused by the aster yellows virus independently of the oat blue dwarf virus, and is transmitted by the six-spotted leafhopper, Macrosteles fascifrons Stål. In four separate greenhouse tests, with leafhoppers from virus-free stock, aster yellows virus was transmitted from infected asters to barley, flax and asters. Averages of the results of these tests indicated that leaf roll dwarf developed in 69 percent of the barley plants, and aster yellows developed in 62 percent of the flax plants and in 91 percent of the aster plants. In six separate greenhouse tests, with leafhoppers from virus-free stock, the virus was transmitted from barley which had leaf roll dwarf symptoms to asters, flax and barley. Aster yellows developed in an average of 72 percent of the aster plants and 48 percent of the flax plants and leaf roll dwarf developed in 16 percent of the barley plants.

In barley, symptoms appeared gradually and infected plants were distinguishable from healthy checks about 3 weeks after the plants were first exposed to infective leafhoppers. Infected barley plants usually became extremely dwarfed, the internodes failed to elongate, and the leaves curled backwards tightly, enclosing the adaxial surface (Fig. 1). An excessive number of leaves and tillers were formed, the foliage was chlorotic, and the plant died prematurely. With few exceptions these plants did not head. When spikes developed they were deformed, the awns were twisted and the florets were sterile. Transmission of virus from these leaf roll barley plants to asters and flax produced aster yellows.

In limited experiments the virus was not transmitted mechanically, through the soil or by the English grain aphid, Macrosiphum granarium Kirby.

It appears from these experiments that the virus which causes yellows in asters or flax and the virus which causes leaf roll dwarf in barley are identical. To the authors' knowledge, this is the first report of infection of barley or of a grass by the aster yellows virus.

FIGURE 1. Vantage barley, 6 weeks old. Right -- leaf roll dwarf. Left -- healthy.

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[1]Paper No. 4322, Scientific Journal Series, Minnesota Agricultural Experiment Station.

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[3]Banttari, E. E., and M. B. Moore. 1959. The cause and transmission of blue dwarf of oats and of two kinds of dwarfing in barley. (Abst.) Phytopathology 49: 533.

HISTORY OF BLACK SHANK IN GEORGIA FLUE-CURED TOBACCO INCLUDING SPREAD OF THE DISEASE IN 1959[1]

John G. Gaines[2]

Abstract

Although black shank of tobacco has been prevalent in the cigar area of southwest Georgia since 1922, it was reported in the flue-cured area of the State only three times prior to 1955. In each instance the disease disappeared. Beginning in 1955 the incidence of black shank increased and by 1959 it had been observed in at least 26 counties embracing all except one of the major flue-cured tobacco-producing counties in south Georgia. The greatest number of infections were observed in 1959 when there was evidence of wind-borne dissemination.

Although black shank, caused by Phytophthora parasitica Dast. var nicotianae (Breda de Haan) Tucker, was reported on cigar wrapper tobacco in the shade-growing district of southwest Georgia as early as 1915[3] and has continued to be a serious problem there since 1922, the flue-cured area of the State remained almost entirely free of the disease until 1955. Beginning in 1956, according to surveys conducted by J. B. Preston, Georgia Tobacco Extension Agronomist, and by the Georgia Department of Agriculture, there was an abrupt increase in the incidence of black shank on flue-cured tobacco farms, culminating in 1958-59 when the disease was observed on many farms scattered throughout the major portion of the Tobacco Belt.

Shade tobacco is grown near the southern boundary of Grady and Decatur counties bordering the Georgia-Florida State line. The flue-cured belt of south Georgia extends from Emanuel, Jenkins, and Bullock counties in the northeast to Grady and Decatur in the southwest, thus blending into the shade district. Only a limited acreage of flue-cured tobacco is grown in Grady and Decatur counties, and most of this is in the northern portions; but on some farms both shade and flue-cured types are grown. In 1924 black shank was reported in two fields of flue-cured tobacco in southwest Georgia[4]. Since that time it has been observed frequently by growers where shade and flue-cured types were grown on the same or nearby farms. The disease was not observed generally in the northern part of these counties until 1940, and following that date it did not recur annually on the same farms there prior to 1959. In a letter to Plant Disease Survey, O. C. Boyd reported black shank to be prevalent in 1928 on one tobacco farm in Mitchell County, joining the northern boundary of Grady County. The disease was not reported again in Mitchell County until 1956. Crop rotation has been practiced by flue-cured tobacco farmers in that area.

In 1933 several tobacco plants infected by the black shank fungus were observed on two nearby farms, one in Toombs County and one in Tattnall County. The disease was observed again on one Ware County farm in 1947. These locations were in the eastern part of the flue-cured tobacco area, far removed from the shade district. On each of these farms the grower located the tobacco bed and field in a new place for several years. When tobacco was grown again in the infested fields, the disease did not recur. It was not observed again in these localities until 1958-59. At no other time prior to 1955 was black shank observed in Georgia flue-cured tobacco outside Grady and Decatur counties.

In 1955 the disease was observed on one farm in Thomas County, bordering the shade district, and on one in Coffee County, approximately 100 miles east of Thomas. In each instance the grower located his tobacco field in a new place the following year and planted a resistant variety. Black shank did not recur on these or adjoining farms prior to 1959.

In 1956 black shank was observed in several locations, but again only on farms where there was no previous history of the disease. In that year only one or two farms were affected in

[1]Cooperative investigation by United States Department of Agriculture, Agricultural Research Service, Crops Research Division and Georgia Coastal Plain Experiment Station, University of Georgia College of Agriculture.

[2]Plant Pathologist, United States Department of Agriculture, Agricultural Research Service, Crops Research Division, Georgia Coastal Plain Experiment Station, Tifton, Georgia.

[3]Tisdale, W. B. 1922. Tobacco diseases in Gadsden County in 1922, with suggestions for their prevention and control. University of Florida Agr. Exp. Sta. Bull. 166: 77-118.

[4]Tisdale, W. B., and J. G. Kelley. 1926. A Phytophthora disease of tobacco. University of Florida Agr. Exp. Sta. Bull. 179: 158-218.

Appling County near the eastern boundary of the belt, and in Colquitt, Mitchell, and Cook counties situated northeast of the shade district. As with earlier infections, severe losses due to disease did not occur on these farms. Tobacco was not grown in the infested fields during 1957-58, and the disease did not recur on those farms during that period.

No black shank was reported in Georgia flue-cured tobacco outside the shade district in 1957. But in 1958 the disease was reported on an increasing number of farms, again only in locations where no black shank had been observed previously. In that year five additional counties were added to the list of infested areas. These were Worth, Lanier, Bacon, Montgomery, and Bullock. The disease was observed at this time in Tattnall County, where a few infected plants were seen in 1933. Thus, by the end of 1958 black shank had been reported at one time or another in at least 14 counties outside the shade district, although the disease did not recur in all these counties in 1958. Until 1956 only a limited number of plants were affected in most of the infested fields and losses were not heavy, but beginning in 1958 measurable losses were sustained in a number of the infested fields, particularly where the disease developed early and subsequent rains caused washing across the rows. Here the disease killed a large percentage of the plants in low places and along the drainage areas.

There were more reports of black shank in 1959 than in any previous year. It was observed in 12 additional counties with no previous black shank history and on at least 75 farms throughout the tobacco-producing areas, with many more unconfirmed reports. The majority of affected fields were in the eastern part of the belt. In nearly all instances the disease appeared in fields with no history of black shank and losses were not heavy; but on a few farms where a susceptible variety was grown on previously infested land, severe disease developed over entire fields and caused almost total losses. As observed in 1958, information obtained from growers concerning the source of tobacco plants indicated much of the inoculum was introduced into fields on plants from affected beds. On other farms, however, infections in previously non-infested fields could not be traced directly to plantbed origin.

Through 1959 black shank had been reported in a total of 26 counties outside the shade district, this number including all except one of the major flue-cured tobacco growing counties of the State. Over the years there has been no indication of a sustained and progressive spread of black shank from the shade into the flue-cured area. At the present time no single known explanation can account for the observed pattern of disease development and recurrence. However, a number of recent departures from the normal in grower practices, together with a recent shift in seasonal distribution of rainfall, could have influenced the sudden upsurge of disease in 1958-59.

Six of these departures from the normal are noted here: 1) Each succeeding year since the unprecedented drought of 1954 has been accompanied by adequate or excessive rainfall during the first half of the growing season, particularly during May, which normally is a dry month. Heavy rains that resulted in some crop damage fell in many localities during these years. The May and early June rains were accompanied by overcast skies and increased atmospheric moisture during the period when the tobacco was cultivated most frequently. Not all parts of the flue-cured area were affected thus, but the changed weather pattern occurred over large sections of the belt. For example, May and June were dry months at Tifton in 1955 but rains were adequate in other sections. Each year during the 4-year period 1956-59 inclusive, the May rainfall at Tifton exceeded 4 inches, while that much rain in May was recorded only four times during the preceding 30 years. 2) After 1954 many tobacco growers practiced irrigation regularly or whenever a temporary deficiency in rainfall was indicated. Irrigation water was taken from ponds and streams. Very little irrigating was done before 1954. 3) With the general adoption of soil fumigation, many growers have discontinued crop rotations for root-knot control and have planted tobacco more often than usual on the same land each year. In addition, the highly susceptible 402 variety has been grown on many of these farms. 4) In recent years an increasing number of growers have bought their tobacco plants from sources outside the Tobacco Belt, particularly from areas south of the Georgia-Florida Tobacco Belt where tomatoes are grown extensively. 5) There also has been an increase in the number of tobacco farmers who grow tomatoes for market, with tomatoes often being grown in the same rotation and field with tobacco. 6) There has been an increase in the use of different kinds of composts and dehydrated manures under tobacco, without due consideration of sources and possible contamination with tobacco and other crop wastes that might harbor the fungus that causes black shank.

The fact that the disease has continued to develop in new areas each year suggests that the fungus has been introduced frequently from unknown sources. Where crop rotations are practiced, symptoms of black shank might not be detected until several years after the introduction;

but, since over 90 percent of the Georgia flue-cured tobacco acreage has been planted in black shank-susceptible varieties, the fungus has had ample host plants on which to develop.

Black shank was observed for the first time at the Georgia Coastal Plain Experiment Station in 1959. It first developed in a plantbed that had been sterilized with 8 pounds of methyl bromide gas per 100 square yards and seeded with the 402 variety. No symptoms of black shank were apparent at the time of transplanting, and the disease was not detected until after the plants were set in the field. Apparently only the plants in one small area of this bed were affected before the first week in April, when transplanting was begun. The source of the inoculum in this seedbed could not be determined. Several other plantbeds were at the same location, but disease symptoms were not observed in these until after advanced stages of black shank were evident in the affected bed. Observations made in the beds and on transplants in the field indicated that the other beds at this location did not become affected until after transplanting was completed.

Since some of the crop rotation, soil fumigation, and other field experiments were set with tobacco plants taken from non-infested beds and others from the infested bed, an opportunity was provided to observe progressive development and spread of black shank during the growing season. By accident, different equipment and laborers were employed to set plants from the non-infested beds; hence the chances for contamination due to handling while transplanting were minimized.

All field experiments set with plants from the affected bed showed occasional black shank symptoms within 30 days after transplanting. The roots and underground stem of the infected plants were darkened and killed before the tops died. Black shank did not develop in fields set with plants from the non-infested beds until after May 20, when a few scattered leaf lesions occurred. Here the fungus entered the plants through the leaves, killing the stalk before the roots were affected and indicating more immediate sources of inoculum than infested beds.

Climatic conditions at Tifton, Georgia were ideal for development and spread of black shank during the 23-day period from May 20 to June 11, when there were 16 days of rain amounting to 6.91 inches. The weather remained cloudy and humid with only two clear days out of 23. Incubator-like conditions prevailed throughout the last 11 days in May, when Phytophthora sporangia developed freely on the lower surface of infected leaves, particularly underneath diseased midribs. During this period large lesions developed on the top leaves of plants two or more feet high. Only one lesion was observed on any single leaf or plant, and often the leaf was infected near the tip end before full size was reached.

In each case the fungus entered the stalk through the midrib of a leaf, thus killing the top well aboveground before the roots or underground part of the stalk were affected. At this time leaf lesions were found almost exclusively near the top of the plant rather than on lower leaves, and they appeared in some but not all experiments set with 402 plants taken from non-infested beds. In certain experiments where leaf lesions developed on plants that originated from such beds, there was a distance of 300 feet or more from infested plots. There was no way spores could have been carried by water into the non-infested fields and splashed onto the top leaves. Since separate equipment and laborers were employed in the different fields, there was no way the fungus could have been carried from the infested to the non-infested areas. Although only a small percentage of plants were thus affected, the leaf spots were scattered more or less uniformly over some field experiments. After the middle of June when atmospheric conditions were less humid and rains less frequent, leaf lesions were confined entirely to the lower leaves, and the spots usually developed close to the stalk rather than near the leaf tip. Also, these late appearing spots usually occurred on plants adjacent to a diseased stalk in the same row. Such infections could readily arise from spores being splashed onto nearby leaves. Often the fungus spreads in this way from one plant to another along the row; but, due to dry weather after midseason, rarely were more than 5 or 6 plants in succession killed at any one place in the row.

One surprising development at Tifton was that black shank was confined almost entirely to the 402 variety, even when other varieties were set in adjoining plots or in the same row with infected 402 plants. The large leaf lesions observed near the top of plants in late May were confined to 402 and certain root-knot resistant breeding lines. Later in the season, lesions on ground leaves developed also on a few plants of Va. 21 and Va. 12. However, on farms out in the State leaf lesions and typical black shank symptoms appeared on all varieties commonly grown there.

Appearance of large leaf lesions in experiments set with plants of the 402 variety taken from non-infested beds followed a set pattern in relation to prevailing wind direction and to location of experiments set with diseased plants. During the last 12 days in May prevailing winds

at Tifton were from the south and east. Occasional leaf lesions occurred throughout experiments set with non-infected 402 plants when they were located 300 to 800 feet north or west of diseased plants, even though adequate barriers and other precautions were provided to prevent contamination. But, when non-infected 402 plants were set on the south and east side of infested plots and the same precautions followed, no black shank developed on the 402 plants at this time or at any later time during the 1959 growing season.

Thus, it was apparent that the disease spread in the direction of prevailing winds. Since barriers existed between experiments to prevent contamination by drainage water, and since the fields were cultivated by different laborers using different equipment prior to the end of May, it was concluded that wind-borne dissemination occurred for distances up to 800 feet. These observations confirm the conclusion of Smith[5] that wind-borne spread of black shank sometimes occurs. Observations at Tifton in 1959 indicated that atmospheric conditions there were suitable for such dissemination for only a very short period during the growing season. It is possible that this explains why the highly susceptible 402 variety developed the large characteristic leaf lesions, while other susceptible but slightly more tolerant varieties momentarily escaped infection.

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[5]Smith, T. E. 1947. Spread of tobacco black shank. Plant Disease Reptr. 31: 120.

OCCURRENCE OF CERTAIN PLANT DISEASES IN KENTUCKY IN 1959[1]

E. M. Johnson, R. A. Chapman and W. D. Valleau

Low temperatures and low rainfall occurred throughout the State during spring planting and the early growing season. Dry weather continued until mid-June or later and low temperatures, especially at night, until well into July. Rains were heavy in late summer, and then the weather was dry again in early fall.

TOBACCO PLANT BED DISEASES

WILDFIRE (Pseudomonas tabaci (Wolf & Foster) Stevens) was less prevalent in tobacco beds than last season when the amount was low. The extensive use of wildfire-resistant varieties probably accounts for less wildfire in tobacco beds.

ANGULAR LEAF SPOT (Pseudomonas angulata (Fromme & Murray) Holland) again appeared in a few tobacco plant beds after having been absent for many years.

ANTHRACNOSE (Colletotrichum destructivum O'Gara) was present in many tobacco beds throughout the State. This tobacco pathogen appears to be the same as the one on legumes and grasses. The improvement of legume and grass pastures in the State may account for the increase in the last few years of anthracnose in tobacco. There was good evidence in some beds that water washing across them had introduced the fungus; in other beds it appeared that the inoculum originated in a grass-legume sod turned under without sufficient soil sterilization to destroy the fungus; and in other cases it appeared that a man walking through a wet pasture and then across the bed may have introduced the pathogen.

COLD INJURY, as in most years, was present in most tobacco beds but appeared to cause little damage to tobacco seedlings.

BLUE MOLD (Peronospora tabacina Adam) appeared in a few beds just prior to when, but more often after, the crop was set. Very mild symptoms consisting of small, dead flecks were observed by a few growers who attributed the trouble to angular leaf spot.

FIELD DISEASES OF TOBACCO

PYTHIUM SOFT ROT (Pythium spp.) occurred in a few fields following setting, but there was less than last year.

BLACK SHANK (Phytophthora parasitica Dast. var. nicotianae (Breda de Haan) Tucker) occurred in areas throughout the State from 2 weeks after setting until cutting time and on many farms with no previous history of the disease. Introduction of the disease by means other than by water from contaminated streams was indicated in some cases. Because of drouth in some areas Burley 11A and 11B developed more black shank than last year.

ANTHRACNOSE was reported as a field disease in tobacco in Kentucky for the first time in 1958. The disease again developed in many fields soon after setting and alarmed some growers. A few weeks after setting plants were badly spotted with lower leaves and some stems covered with irregular dead spots 1/8 to 1/4 inch in diameter. Dead lens-shaped depressed spots, present on the under sides of midribs, often caused midveins to break. Acervuli with setae and spores were present in leaf spots and in necrotic spots on stems and midribs or they developed overnight when necrotic tissue was placed in moist chambers. By early July, affected plants had almost completely recovered and there appeared to be little or no damage caused by the disease.

MANGANESE TOXICITY Much burley tobacco is grown in continuous culture with a winter cover crop of grain or grain and vetch and extremely large amounts of mixed commercial fertilizer and ammonium nitrate are applied every year. Under these conditions areas of some fields become very acid and manganese toxicity develops in the leaves of tobacco. In 1959 manganese toxicity occurred on soils with pH 4.5 to 4.8. Leaves of affected plants contained 1780 to 5350 parts per million of manganese.

MANURE INJURY For several years specimens of tobacco failing to make any growth from setting until mid-July have been received. No black root rot fungus, nematodes or other pathogens were found by microscopic examination. In 1959 an unusual number of specimens were re-

[1] The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published with approval of the Director.

ceived. In late June and early July fields in the central area showing uneven growth were studied. In some areas of these fields severely affected plants appeared smaller than when set, while in other areas of the same field the plants were knee high. The original root system of dwarfed plants was dead or inactive, and new roots that had developed had died. In every case studied heavy applications of strawy barn manure, and in one case strawy turkey manure, had been used. When the soils were examined much undecomposed strawy material was found. It is believed that because of low temperatures and low moisture in May that decay of the cover crop and strawy manure had not occurred normally. Decaying cover crops sometimes produce toxic materials that stunt or delay the growth of tobacco seedlings (1). This toxic effect may have been aggravated by the heavy application of strawy manure. Small affected seedlings set in pots of greenhouse soil sent out new roots and after a time grew normally, indicating that the stunting, in most cases, may be temporary. The grower who used large amounts of strawy turkey manure stated that at cutting time the tobacco in the manured areas was half the size of that in areas of the field receiving no manure.

FROGEYE (Cercospora nicotianae Ell. & Ev.) occurred exceptionally early in young tobacco and in destructive amounts in mature tobacco. In many fields, during a rainy period half or more of the lower leaves were so completely covered with frogeye spots that they were destroyed. Brown soft, rotted areas occurring at the base of petioles of affected leaves caused them to slough off. Motile bacteria that rotted carrot slices were present in the rotted petioles. Typical frogeye spots were numerous on all leaves to the top of the plants and where suckers were present these were spotted. As a result of frogeye, leaves that ordinarily would have gone into lug grades were graded as flyings. Graders and warehousemen said there was a marked scarcity of sound lugs and that there was more frogeye in all grades of tobacco than most of them had ever seen. This greatly increased incidence of frogeye caused greater damage than at any time since the 1920 outbreak. Many growers attributed both epidemics to wildfire. All varieties appeared susceptible.

WILDFIRE and ANGULAR LEAF SPOT were of minor importance in tobacco; however, the latter appears to be on the increase after an almost complete absence of many years. In some fields Burley 21, previously reported as being resistant to angular leaf spot, was damaged late in the season by this disease.

BLACK ROOT ROT (Thielaviopsis basicola (Berk. & Br.) Ferr.) again caused considerable damage to varieties of low resistance because of the cool weather that lasted, especially at night until well into July. The disease often damaged varieties Ky 16, Ky 41A and Burley 21.

ETCH appears to be increasing in several areas of the State. The disease caused damage in several fields in the central part of the State.

CURLY TOP and CLUB ROOT No specimens of either of these virus diseases of tobacco were received during the year.

ROOT KNOT (Meloidogyne incognita (Kofoid & White) Chitwood) was found in one tobacco field each in Russell and Woodford counties. Both were former garden areas. Typical galls were present on the roots and the plants made very poor growth.

TOMATO

TOBACCO MOSAIC is perhaps the main cause of low yields of tomato in plastic greenhouses and in many fields. In many houses and fields all plants are often affected when they reach bearing size. Plastic houses free of mosaic occur but they are rare. Inoculations to tobacco indicate that there are many strains of tobacco mosaic affecting tomato which differ from the usual strains of tobacco mosaic.

EARLY BLIGHT (Alternaria solani (Ell. & G. Martin) Sor.) and LATE BLIGHT (Phytophthora infestans (Mont.) D By.) were found in a few plastic greenhouses. One house of tomatoes, started while there were still tomatoes outside, was completely destroyed by late blight.

VERTICILLIUM WILT (Verticillium albo-atrum Reinke & Berth.), identified by isolation, was destructive to tomato in some plastic greenhouses and was identified by isolation in two fields.

FUSARIUM WILT (Fusarium oxysporum f. lycopersici Snyder & Hansen) was identified by isolation in one plastic house of tomatoes.

LEAF MOLD (Cladosporium fulvum Cke.) was present in varying amounts in most plastic greenhouses. Damage was greater in poorly ventilated houses and in those where fertility was low. Leaf mold appeared in some outdoor plantings late in the season but appeared to cause little damage.

CURLY TOP A tomato plant with crinkled leaves with downward rolled leaf margins and

downward bent petioles was received from an outdoor planting in northern Kentucky. The plant was set in the greenhouse and is still alive after 8 months. Small fruits which ripened normally were produced. Seed from ripe fruit was sowed at two different times. The resulting seedlings appeared entirely normal. The virus disease was transmitted by grafting to tomato where the symptoms were like those in the original field plant. The virus was also transmitted by grafting to tobacco plants which developed pale narrow, twisted leaves with downward rolling margins and downward bent petioles similar to tobacco plants in the field affected with curly top. Mechanical inoculations to tomato and tobacco were negative. The symptoms in tomato are not entirely like the symptoms of curly top in published descriptions, but apparently symptoms in tomato can vary greatly depending on the strain of the curly top virus.

ROOT KNOT (Meloidogyne incognita) was present and destructive in some plastic greenhouses.

HORMONE INJURY In one plastic greenhouse where a fruit setting hormone spray had been used oftener than recommended, the leaves of all plants developed distortion indistinguishable from 2,4-D injury. When tomato seedlings in the greenhouse were sprayed with a higher than recommended concentration of the same hormone they developed distortion like that resulting from minute amounts of 2,4-D.

ELM

DUTCH ELM DISEASE (Ceratocystis ulmi Buis.) was identified by isolation of the fungus from two trees in Paducah. This is about 50 miles west of infected trees found at Princeton in 1958.

CORN

In a river bottom area in Franklin County, hybrid field corn broke over at the first or second node. These nodal areas were brown with discoloration extending into the pith. The corn remained green until the stalks were almost rotted through. A Phycomycete was present in the discolored tissue. This appears to be the Pythium disease that was reported several years ago from a river bottom area west of Louisville (2).

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KENTUCKY AGRICULTURAL EXPERIMENT STATION, LEXINGTON

DUTCH ELM DISEASE IN KANSAS IN 1959

C. L. Kramer[1], and Hugh E. Thompson[2]

During the past year Dutch elm disease (Ceratocystis ulmi) has continued its advance into Kansas. It has become widespread in the Kansas City area of Wyandotte and Johnson counties where it has been known since 1957 (Fig. 1). New locations have also occurred in Leavenworth and Miami counties where the disease was first reported in 1958. In addition to these records, five new counties have been added to the list from which positive locations of the disease are known (Fig. 1). These new locations include one in Ottawa, Franklin Co.; three in Lawrence, Douglas Co.; one in Emporia, Lyon Co.; two in Coffeyville, two in Independence and one in Caney, Montgomery Co.; and one in Galena, Cherokee Co. Although some 30 collections were made from suspected trees in the counties of east-central Kansas separating the known areas of infection in the north from those of Montgomery and Cherokee counties in southern Kansas, none was positive. Collections were made by Hugh E. Thompson and LaVerne Calkins[3], while isolations and identifications were made by C. L. Kramer and some by Robert Lichtwardt[4].

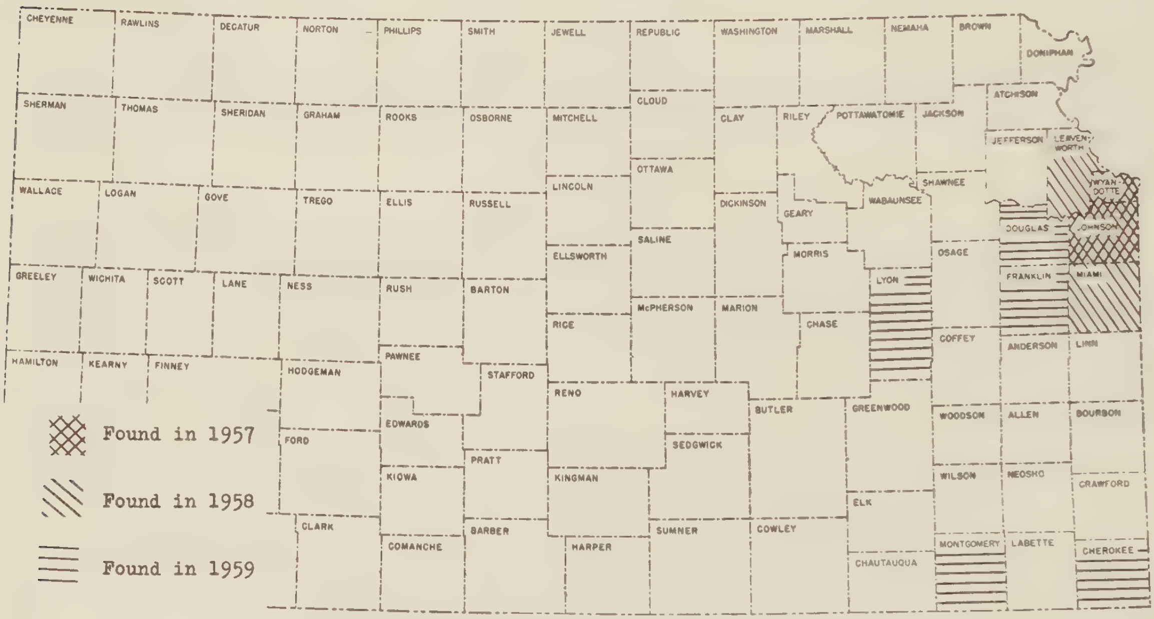

FIGURE 1. Distribution of Dutch elm disease in Kansas, 1959.

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THE STATUS OF DUTCH ELM DISEASE IN ILLINOIS

Dan Neely, J. C. Carter[1], and R. J. Campana[2]

Abstract

Presence of the Dutch elm disease fungus, Ceratocystis ulmi (Buis.) Moreau, has been confirmed in all 102 counties of Illinois. In the cities of Champaign, Urbana, and Bloomington over 80 percent of the elms have been killed by Dutch elm disease and elm phloem necrosis. In a number of cities in northern Illinois that have been conducting comprehensive disease control programs for four or more years, annual Dutch elm disease losses have been kept below 1 percent of the elm populations.

The increase in extent and severity of Dutch elm disease (causal fungus, Ceratocystis ulmi) in Illinois, following the first report of its presence in 1950 by Carter (5), in the years 1950 through 1956 has been presented by Campana (1, 2) and Campana and Carter (3, 4). By 1954 Dutch elm disease had spread extensively throughout the southern half and the northeastern quarter of the State. The disease continued to spread, moving into areas in northern and western Illinois and by 1956 the presence of the disease had been confirmed by laboratory culture in 86 of the 102 counties of the State.

In 1957, 1958, and 1959 Dutch elm disease continued to spread westward toward the Illinois-Iowa boundary (Fig. 1). Spread of the disease to eight additional counties in 1957 was confirmed by laboratory culture. These counties were Brown, Bureau, Cass, Fulton, Henry, Rock Island, Stark, and Stephenson. In 1958 the disease was found in five more counties: Hancock, Henderson, Mason, McDonough, and Warren. The presence of Dutch elm disease was confirmed in the remaining three counties of the State, Carroll, Jo Daviess, and Mercer, in 1959. Within 10 years after the discovery of the first diseased tree, Dutch elm disease has spread to all 102 counties of the State.

Since 1956, it has not been possible to determine the number of trees or the number of localities affected by Dutch elm disease within the State. In Bloomington, Champaign-Urbana, Kankakee, and Rockford, the disease reached an epiphytotic level prior to 1957 and has continued to kill large numbers of elms annually. The disease has reached the epiphytotic stage of development in Dwight, Streator, and Pontiac only during the past 3 years. Dutch elm disease takes a heavy toll of elms in southern Illinois but receives less attention in that region because of the earlier appearance and rapid spread of elm phloem necrosis, which had killed many thousands of elms before Dutch elm disease invaded the area.

In Champaign-Urbana, where a careful survey of the elm population has been conducted annually since 1944, losses during the last 5 years due to Dutch elm disease have been high (Table 1). Based upon an estimate of the elm population in 1951 of 15,196 trees, the percentage of elms killed each year by Dutch elm disease and by phloem necrosis was calculated. The percent of the elms which remained each year that were killed by Dutch elm disease and by phloem necrosis also was calculated. The data in Table 1 show that 10,201 trees or 67.1 percent of the original elm population have been killed by Dutch elm disease, while only 2952 trees or 19.4 percent of the elms have been killed by phloem necrosis. The present residual elm population is approximately 2040 in the two cities. The percent of the residual elm population affected by Dutch elm disease has substantially increased each year since the first occurrence of this disease in 1951.

Surveys of the elm population of Bloomington have been taken since 1948. The number of elms killed each year by Dutch elm disease is recorded in Table 2. Annual phloem necrosis losses are given since 1953. In conjunction with the disease survey in 1959, a tally was made of the surviving elms. The number of surviving elms and the number of known diseased elms gave an original elm population of approximately 9055 trees. This number includes those elms planted since the survey began. The percentage losses due to Dutch elm disease and to phloem necrosis based on the original elm population and the remaining annual elm population are included in Table 2. At present 80.1 percent of the elms in Bloomington have been killed by the two diseases; 35.0 percent by phloem necrosis and 45.1 percent by Dutch elm disease.

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FIGURE 1. Known distribution of Dutch elm disease in Illinois from 1956 to 1959, indicating spread each year by counties.

Over 50 cities in northern and northeastern Illinois are now conducting community-wide programs for the control of Dutch elm disease. They are utilizing comprehensive sanitation and spraying control procedures to keep disease losses to a low level. These cities are located in areas where elm phloem necrosis is not known to occur.

Questionnaires prepared by the Natural History Survey concerning the application of control practices and the effectiveness of disease control were mailed to 52 Illinois cities. Thirty-seven of these questionnaires were completed and returned by municipal officials responsible for Dutch elm disease control programs. The following information is based upon information obtained from these questionnaires and from personal conversations with many of the municipal officials.

Table 1. Incidence of Dutch elm disease and elm phloem necrosis in Champaign-Urbana.

Year	Number of trees affected		Per cent of original population		Per cent of residual population	
	Dutch elm disease	phloem necrosis	Dutch elm disease	phloem necrosis	Dutch elm disease	phloem necrosis
1944-50	0	428	0	2.8	0	
1951	1	359	.01	2.4	.01	2.4
1952	11	555	.07	3.6	.08	3.9
1953	164	388	1.1	2.6	1.2	2.8
1954	694	179	4.6	1.2	5.2	1.3
1955	1805	123	11.9	0.8	14.5	1.0
1956	1836	60	12.1	0.4	17.5	0.6
1957	2116	368	13.9	2.4	24.6	4.3
1958	1770	344	11.6	2.3	29.0	5.6
1959	1804	148	11.8	0.9	45.1	3.7
Total	10201	2952	67.1	19.4		

Table 2. Incidence of Dutch elm disease and elm phloem necrosis in Bloomington.

Year	Number of trees affected		Per cent of original population		Per cent of residual population	
	Dutch elm disease	phloem necrosis	Dutch elm disease	phloem necrosis	Dutch elm disease	phloem necrosis
1948-53	0	932	0	10.3	0	
1954	10	222	0.1	2.5	0.1	2.7
1955	242	234	2.7	2.6	3.1	3.0
1956	517	400	5.7	4.4	7.0	5.4
1957	894	520	9.9	5.7	13.8	8.0
1958	1424	761	15.7	8.4	28.0	15.0
1959	997	100	11.0	1.1	34.4	3.5
Total	4084	3169	45.1	35.0		

Table 3. Percent of original elm population lost because of Dutch elm disease in Illinois cities that have had community-wide control programs for at least four years.

City	Year Program Began	Original Elm Population	Per cent of original population			
			1956	1957	1958	1959
Batavia	1956	6,230	1.56	2.17	3.58	2.60
Chicago (forestry & parkways)	1955	300,000	.01	.03	.11	.16
Clarendon Hills	1955	6,000	.01	.03	.07	.20
Evanston	1956	17,920[a]	.11	.15	.46	.49
Geneva	1956	4,432[b]	1.04	1.04	1.31	1.17
Glencoe	1955	5,500	.55	.49	.45	.33
Glenview	1956	7,689	----	.35	.34	.26
Hinsdale	1955	11,050	.05	.51	.29	.28
Kenilworth	1956	5,002	.14	.18	.36	.24
Lincolnwood	1955	11,800	.09	.11	.15	.09
Mount Prospect	1955	8,100	.05	.09	.11	.14
Riverside	1955	4,500	----	.15	.27	.15
Western Springs	1955	3,573[a]	.11	.25	.28	.33
Winnetka	1956	6,700	.31	.32	.31	.20

[a]Trees on public property only.
[b]Trees on parkway only.

All of the cities with complete and comprehensive Dutch elm disease control programs are following closely the recommendations compiled by the Specifications Committee of the Midwestern Chapter, National Shade Tree Conference and printed as a Guide for Community-Wide Control of Dutch Elm Disease. This publication describes in detail the various aspects of surveying, scouting, sanitation, and spraying. The purpose for each is explained.

Surveys in many of the cities with disease control programs to determine the size, condition, and location of the elm population to be protected from Dutch elm disease and to locate for elimination all elm wood that might serve as a potential hazard for bark beetle breeding are incomplete. In only five of the 37 reporting cities has a complete tally of all publicly and privately owned elms in the control areas been made. In 19 cities elms on parkways and in public parks were surveyed but elms on private property were not surveyed. Complete surveys have not been attempted in the other cities, but the condition of the elm population in each city was noted in conjunction with routine scouting for Dutch elm disease.

Scouting to detect trees with current Dutch elm disease symptoms and to locate other elm material that might serve as potential sources of elm bark beetle breeding is an essential part of a successful disease control program. In 28 of the reporting cities the scouting is conducted by, or under the direct supervision of, a trained forester or experienced arborist. In each of these cities a block-by-block survey of the entire tree population is completed at least twice during the summer.

Sanitation practices to eliminate actual or potential sources of elm bark beetles is a basic control procedure and is rigidly conducted in all those cities with successful disease control programs. Twenty-seven cities have local ordinances to enforce removal of known diseased trees from private property, so they have been able to obtain prompt removal and destruction of diseased trees on both public and private lands. In the other reporting cities there has been no need for an ordinance because of the favorable response to the program by the citizens. Sanitation pruning of healthy elms has been started or has been completed in 21 cities.

Spraying healthy elms with an insecticide to prevent feeding by elm bark beetles is recommended for all elms but has been practiced principally upon publicly owned trees. The 37 reporting cities state that all parkway elms are sprayed annually during the dormant season. In only four cities are all publicly and privately owned elms sprayed. The single application of an insecticide during the dormant season is the spray schedule that is chiefly followed. Only four of the cities report continued use of the foliar spray. Bird mortality was stated to be a major problem in six cities where DDT was the insecticide used. Only one city is now using methoxychlor in place of DDT. One city sprays with DDT in the fall and with methoxychlor in the spring. Sprayers of the mist blower type are used in 28 cities, hydraulic sprayers are used in eight cities, and both types of sprayers are used in one city.

A majority of the cities with control programs are keeping new infections below 1 percent annually of the elm tree population. In neighboring cities without control programs or with incomplete control programs, the number of new infections increases annually. Illinois cities reporting on Dutch elm disease control programs which have been in effect for at least 4 years are listed in Table 3 and the percent of elms lost annually because of Dutch elm disease is given. There are numerous other cities which have well-organized and well-executed Dutch elm disease control programs in the State that have been in effect less than 4 years. Many of these cities have obtained similar results.

Those cities in Illinois that are successfully controlling Dutch elm disease find that the cost of sanitation and spray control procedures is less than the cost of removing the trees that would be lost if the disease should reach an epiphytotic level. Through control practices they are able to help maintain the beauty of their cities by preserving the elms.

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CONTROL OF CELERY EARLY BLIGHT AND BACTERIAL BLIGHT IN THE EVERGLADES WITH DYRENE COMBINATIONS

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Abstract

Dyrene used alone at 1 1/2 pounds/100 gallons controlled celery early blight. Dyrene was effective at 1 pound/100 gallons when combined with either tribasic copper sulfate or copper zinc chromate at 2 pounds/100 gallons. Dyrene added to the copper spray did not greatly affect bacterial blight control. Dyrene caused no visible toxicity to celery plants in the seed-bed or field whether used alone or with copper and/or streptomycin.

The serious foliage diseases of celery in the Everglades are early blight, caused by *Cercospora apii* Fres., and bacterial blight, caused by *Pseudomonas apii* Jagger. Early blight is by far the more common; it is usually present throughout the year in every field and seed-bed. Bacterial blight is more difficult to control but is usually serious only during the warmer months from late spring to early fall. One or both of these diseases may become the limiting factor in field celery production during the late spring.

Dyrene (50% 2, 4-dichloro-6-(*o*-chloroanilino)-s-triazine) is the only commercially available fungicide which has promise of competing with the dithiocarbamates for control of celery early blight. In fungicide trials of Cox (1), Darby (2), Wilson (6) and Van Nostran (5) Dyrene has controlled early blight as well as or better than the carbamates. In addition to control of early blight, Dyrene has been reported effective against Rhizoctonia rot (1) and late blight (3, 6) of celery. However, a serious objection to the use of Dyrene has been its high cost. This objection has been partially removed by the finding of Cox (1) and Van Nostran (5) that Dyrene and tribasic copper sulfate when mixed in the spray tank are additive in control of early blight. This additive effect is more important in view of the fact that tribasic copper sulfate offers some control of bacterial blight.

Tests reported here were initiated to determine minimal amounts of Dyrene and copper which could be used for celery disease control and to determine whether Dyrene can be used on celery seed-beds without causing damage to tender seedlings.

MATERIALS AND METHODS

Celery variety Utah 52-70 was used in all tests. Plants were sprayed with a small plot sprayer at 300 to 400 pounds p.s.i. at 4-to 5-day intervals. Parathion was used as necessary for insect control. Disease severity was scored by percentage of leaf surface blighted and given a numerical rating from 1 to 12 according to the Horsfall and Barratt system (4).

RESULTS OF FIELD TESTS

Twenty-five-foot single row plots were used in a random block design with five replications. Gallons per acre of spray ranged from 50 to 150 with two nozzles per row on young celery and increasing to six nozzles on mature plants.

The first test compared three concentrations of Dyrene and two of tribasic copper sulfate alone and in combinations on control of early blight and bacterial blight. Results are recorded in Table 1. The additive effect of Dyrene and copper on early blight control was demonstrated in this experiment, but not at all concentrations. Dyrene at 1 1/2 pounds/100 gallons was sufficiently effective in controlling early blight so that the addition of copper did not improve control. The additive effect was in evidence with Dyrene at 1 pound/100 gallons plus copper at either 2 pounds/100 gallons or 4 pounds/100 gallons. Dyrene at 1/2 pound/100 gallons plus copper at 4 pounds / 100 gallons was additive, but not Dyrene 1/2 pound / 100 gallons plus copper at 2 pounds / 100 gallons. Although plants were rated for bacterial blight on two occasions, the second ratings could be made only on those plots with good early blight control. Under the conditions of light bacterial blight infestation in this test tribasic copper sulfate reduced severity of this disease.

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[1] Assistant Plant Pathologist. Florida Agricultural Experiment Station Journal Series, No. 1065.

Table 1. Effectiveness of spray treatments using various concentrations of Dyrene and tribasic copper sulfate (TBCS) alone and in combination on early blight and bacterial blight of field celery.

Treatment	Concentration per 100 gallons (in pounds)	Early blight rating[a]		Bacterial blight rating		Yield in pounds per 25-foot plot
		Nov. 17	Dec. 1	Oct. 14	Dec. 1	
Dyrene	1/2	3.8	4.5	3.1		57
Dyrene	1	2.7	3.4	3.2	3.0	65
Dyrene	1 1/2	2.2	2.7	3.3	2.8	67
TBCS	2	5.7	5.9	3.0		43
TBCS	4	5.3	6.2	2.5		42
Dyrene plus TBCS	1/2, 2	3.5	4.0	2.4	2.4	66
Dyrene plus TBCS	1/2, 4	3.0	3.5	2.2	1.7	72
Dyrene plus TBCS	1, 2	2.6	2.7	2.3	2.4	76
Dyrene plus TBCS	1, 4	2.3	2.6	2.3	2.1	78
Dyrene plus TBCS	1 1/2, 2	2.2	2.2	2.0	2.3	72
Dyrene plus TBCS	1 1/2, 4	1.8	2.3	1.8	1.7	76
Check		6.8	7.2	3.8		19
LSD 0.05		0.5	0.6	0.4	0.5	8

[a]Horsfall and Barratt's disease rating system: 1 = no disease and 12 = plants killed.

Table 2. Effectiveness of spray treatments on field celery for control of early blight.

Treatment[a]	Concentration per 100 gallons or parts per million	Early blight rating[b]			Yield in pounds per 25-foot plot
		April 24	May 4	May 15	
Dyrene + TBCS	1 lb., 4 lb.	1.0	1.4	1.8	107
Dyrene + copper zinc chromate	1 lb., 2 lb.	1.3	1.7	2.0	102
Dyrene + streptomycin	1 lb., 100 ppm	1.2	1.7	2.5	118
Nabam + $ZnSO_4$	2 qt., 3/4 lb.	1.6	2.1	2.9	99
Maneb	1 1/2 lb.	1.5	2.3	3.0	104
Phaltan	2 lb.	1.6	3.1	3.9	113
Captan	2 lb.	2.2	4.2	4.8	90
Cyprex	1 lb.	2.3	4.8	5.6	60
Check	---	3.1	6.4	6.8	65
LSD 0.05		0.4	0.8	0.7	14

[a]Source of materials: copper zinc chromate, Miller 658; streptomycin, Agrimycin 100; nabam, liquid Parzate; maneb, Dithane M-22.
[b]Horsfall and Barratt's disease rating system: 1 = no disease and 12 = plants killed.

The minimal concentration of Dyrene necessary for control of celery early blight is 1 1/2 pounds/100 gallons if used alone; if combined with 2 pounds of tribasic copper sulfate the level of Dyrene can be reduced to 1 pound/100 gallons. The mixture is less expensive than the Dyrene alone. When bacterial blight is a threat, there is some indication that the level of tribasic copper sulfate should be increased to 4 pounds/100 gallons.

The second field test compared Dyrene combined with 1) tribasic copper sulfate, 2) copper zinc chromate, and 3) streptomycin. Several organic fungicides used alone were also included in the trial. Results are presented in Table 2. The Dyrene combinations controlled early blight best followed by nabam-$ZnSO_4$ and maneb. Phaltan, captan, and Cyprex were relatively ineffective in controlling early blight. Copper zinc chromate was as effective in combination with Dyrene as tribasic copper sulfate. Apparently the source of basic copper is not important in the additive effect of the copper-Dyrene mixture on early blight control.

Table 3. Effectiveness of spray treatments on control of early blight of celery in seed-beds.

Treatment[a]	Concentration per 100 gallons or parts per million	Early blight rating[b] Sept. 1	Sept. 8
Dyrene	1 1/2 lb.	1.0	1.0
TBCS	4 lb.	2.2	2.2
Streptomycin	50 ppm	4.7	5.7
Dyrene + streptomycin	1 1/2 lb., 50 ppm	1.0	1.0
TBCS + streptomycin	4 lb., 50 ppm	2.0	2.2
Dyrene + TBCS	1 lb., 4 lb.	1.0	1.2
Dyrene + TBCS + streptomycin	1 1/2 lb., 4 lb., 50 ppm	1.0	1.0
Maneb + streptomycin	1 1/2 lb., 50 ppm	1.0	1.0
Zineb + thiram + streptomycin	2 lb., 1 1/2 lb., 50 ppm	1.0	1.0
Check	---	5.7	6.5
LSD 0.05		1.6	0.4

[a]Source of materials: streptomycin, Agrimycin 100; maneb, Dithane M-22; zineb, Parzate.

[b]Horsfall and Barratt's disease rating system: 1 = no disease and 12 = plants killed.

RESULTS OF SEED-BED TEST

Two 4 x 300-foot seed-beds were used for the trial. There were four replications (two on each seed-bed) of 4 x 10-foot plots arranged in a random block design. Materials were applied with a three nozzle hand boom attached to a power sprayer by a length of rubber hose. Rate of application was 10 to 12 gallons per seed-bed. Results are presented in Table 3. Plants in these seed-beds were in a thinly populated stand which allowed for good air circulation and maximum spray coverage. This probably accounts for the perfect control of early blight obtained with Dyrene, maneb, and zineb plus thiram. Dyrene did not cause any detectable toxicity to the plants even when combined with tribasic copper sulfate and streptomycin. It thus appears safe to use Dyrene on celery at any stage of its growth.

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DWARFING OF SUMMER TOMATOES BY CREASE STEM

P. A. Young[1]

Summary

Crease stem causes dwarfing, short internodes, rigidly upright stems and very compact bunchy appearance of tomato plants. The upper parts of main stems show flattened areas with deep longitudinal creases. Affected tissues often are brown. Crease stem apparently is a physiological abnormality that has been associated with abundant nitrogen in warm wet soil. Tomato varieties and selections ranged from immune to very susceptible to crease stem.

Many selections of summer tomatoes showed high percentages of their plants with crease stem by July 2, 1959 (Fig. 1). This was 1 month after the plants were set in fertile wet soil and a week after the earliest normal plants began to bloom. The abnormal plants were tagged and nearly all of them showed at least one stem per plant with creases (Fig. 2). The 1370

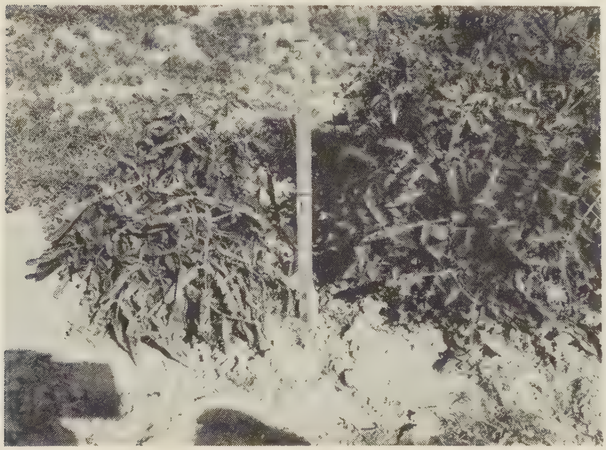

FIGURE 1. Rutgers tomato plants; normal on right and with crease stem on left.

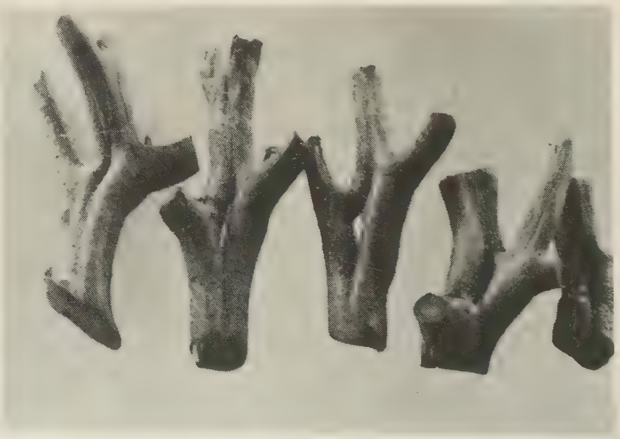

FIGURE 2. Pieces of Rutgers tomato stems showing creases and holes in stems.

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tomato plants of the selections in two fields showed the following percentages with crease stem on July 14th: Rutgers 17%, Greater Baltimore 43%, Hotset 4%, Firesteel 2%, S1948[2] 68%, S1787 43%, S2017 32%, and nine selections of STEP 329[3] 17% to 74%, but no crease stem was found in the plants of S1112, S1447, S1949, CP1951[2], S2012 or CP2064. None of these tomatoes showed any crease stem in the larger spring crop in 1959.

The symptoms of crease stem were dwarfing, short internodes, rigidly upright stems and very compact bunchy appearance (Fig. 1). The thick upper parts of some main stems or branches showed flattened areas with deep longitudinal creases. Some of the creases extended through some Rutgers stems like slit windows (Fig. 2). The creases were shallower and often brown in stems of STEP 329 tomato. Brown discoloration and sometimes hollow spots commonly occurred in the stems near the creases. With few exceptions, the plants that showed creases in their stems also showed the bunchy-top symptoms. Crease stem delayed blooming 1 to 2 weeks in Rutgers but not in STEP 329 tomatoes. The bunchy-top symptoms usually became inconspicuous within a month, due to rapid growth of the plants.

In the spring crop of 1955, V24-49 Rutgers tomatoes showed crease stem in most of its plants growing in fertile wet soil but in none of its plants growing simultaneously in deep sandy soil with low fertility. In 1947 a commercial field of tomatoes showed about one-third of the Rutgers tomatoes with crease stem, but none of the Stokesdale variety. Serious crease stem was studied in two other commercial fields.

The dwarfing symptom of crease stem was described as the short internode abnormality by Young (2). It was named crease stem by Spencer and Geraldson (1) and Young (3).

Crease stem presumably is a physiological abnormality. Near Jacksonville, Texas symptoms of crease stem appeared only in tomatoes growing in fertile warm wet soil, probably containing abundant nitrogen. In 1959 the tomatoes grew in rich, wet, poorly drained Caddo loamy fine sand. Tomato selections apparently range from practical immunity to severe susceptibility to crease stem.

Crease stem can be avoided by raising resistant varieties such as Stokesdale and providing adequate drainage for the soil. Abundant nitrogen fertilizer should be avoided for susceptible tomatoes before they have many fruits larger than 1 inch in diameter.

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[2] The numbers refer to dissimilar selections of tomatoes being tested for their qualities.

[3] STEP 329 refers to the Southern Tomato Exchange Program No. 329.

THE RELATION OF FERTILIZATION TO DEVELOPMENT OF BACTERIAL SPOT ON PEPPER[1]

Jack Taylor and J. W. Dobson, Jr.[2]

Abstract

Observations on pepper fertility tests in 1953 indicated that development of bacterial spot, caused by Xanthomonas vesicatoria, was affected by rates of fertilization. In further studies during 1959 with fertilizer treatments ranging from 0 to 10,000 pounds per acre of 12-12-12 equivalent, bacterial spot incidence decreased as fertilizer rates increased to 5000 pounds per acre.

Bacterial spot, caused by Xanthomonas vesicatoria (Doidge) Dowson, severely damages pepper in the mountains of Georgia during seasons in which frequent showers occur. When the disease develops early in the season, the pepper plants are stunted and relatively nonproductive. Late season infection causes defoliation, which reduces yields and also results in sunscald of fruits. Outbreaks of bacterial spot in 1953 and 1959 were the worst experienced in this area. The disease developed early in 1959 and continued in epiphytotic proportions throughout the season. Many fields were completely destroyed, and production was cut drastically in all fields observed. Serious losses have also been reported in Delaware (3) and Florida (5).

Bacterial spot of pepper has been difficult to control. There are no varieties of sweet pepper with sufficient resistance to the disease. Sprays and dusts are only partially effective and therefore have not been used extensively. Consequently, the use of disease-free seed, seed treatment, and field sanitation have been recommended for the control of bacterial spot, although the inadequacy of these measures is indicated by their failure to control the disease in seasons favorable for its development. Attempts to correlate the source of plants or cropping systems with disease development usually have failed. However, observations on pepper fertilizer experiments in 1953 indicated a correlation between fertilizer rates and development of bacterial spot. Tests initiated in 1959 gave additional information on this relationship.

MATERIALS AND METHODS

The effects of fertilizer treatments on disease development and on plant growth of Yolo Wonder bell pepper were studied on two different soil types during 1959. Both soils were medium to high in fertility. In each test treatments consisted of an unfertilized control and applications of a 4-12-12 fertilizer at 625, 1250, 2500, 5000 and 10,000 pounds per acre. A precision distributor especially designed for plot work was used to apply the fertilizer in a single band (4). After the fertilizer had been distributed in the furrow, soil was listed onto the furrow, and pepper plants were set 15 inches apart in the resulting ridge. Sufficient ammonium nitrate was applied about 30 days after setting the plants to provide a 1:1:1 ratio of N, P_2O_5, and K_2O. Future reference to fertilizer treatments will be in pounds per acre of 12-12-12 fertilizer. The treatments were compared in a randomized block with four replications.

Test No. 1: This test was on Transylvania silt loam (bottom land) and was irrigated with 1 3/4 inches of water 24 hours after planting. Three sprays containing 3/4 pound of Manzate (70 percent) and 2 pounds of Tri-Basic Copper Sulfate (53 percent) in 100 gallons of water were applied uniformly to all plots during July. Data were taken on bacterial spot, Cercospora leaf spot, plant survival, fertilizer toxicity, and yield. An analysis was made of the nitrogen, phosphorus, potassium, and total mineral content of the pepper leaves.

Test No. 2: The soil was Hiwassee clay loam (upland). This test was not irrigated or sprayed, and Cercospora leaf spot was encouraged by distributing diseased leaves throughout the field. Observations were made on bacterial spot, Cercospora leaf spot, plant survival, and fertilizer toxicity.

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Table 1. Defoliation, bacterial spot ratings, and yields of pepper at different rates of fertilization.

Fertilizer applied in pounds/acre: 4-12-12 at planting	Fertilizer applied in pounds/acre: Ammonium nitrate side dressing	Defoliation caused by bacterial spot[a] on August 6: Average/plot	Defoliation caused by bacterial spot[a] on August 6: Average/plant	Bacterial spot ratings[b] on intact leaves on August 6	Yield: Pounds/plot
0	0	392	19.6	4.0	37
625	150	244	12.2	2.7	51
1,250	300	193	9.6	2.8	58
2,500	600	133	6.6	2.0	61
5,000	1,200	26	1.3	1.3	64
10,000	2,400	29	1.4	1.0	40
LSD .05		100			16
.01		142			23

[a]Defoliation based on average number of leaves under 20 plants.

[b]Disease ratings were 0-5 on each plot with 0 = no infection, 1 = trace, 2 = slight, 3 = moderate, 4 = severe, and 5 = very severe with extensive blighting of leaves.

RESULTS

Bacterial spot development was similar in both tests during the early phases. Cercospora leaf spot developed in test No. 2 late in the season. In general, plant growth and fertilizer toxicity symptoms were about the same on both soils.

Test No. 1: Bacterial spot caused extensive leaf infection and defoliation in unfertilized plots during the first 6 to 8 weeks after setting plants in the field (Figs. 1 and 2), but severity decreased as fertilization rates increased up to 5000 pounds per acre (Table 1). Infection was slight at the 5000 and 10,000 pound rates until the latter part of September, and then defoliation was not severe.

Cercospora leaf spot appeared in the test in early July. It developed extensively in unsprayed plots of a nearby spray test, but apparently the three sprays of Manzate and Tri-Basic Copper Sulfate prevented further development of this disease in the fertilizer test without materially affecting development of bacterial leaf spot.

The fertilizer treatments caused noticeable reductions in stands and in growth of plants in some of the plots receiving 10,000 pounds of fertilizer per acre, and marginal leaf chlorosis developed in mature leaves at the two highest rates of fertilization. In general, plant vigor and color were best in plots receiving 1250, 2500 and 5000 pounds of fertilizer per acre. Analysis of leaves for percent nitrogen, phosphoric acid, potash, and total minerals did not show significant differences due to fertilizer rates.

Test No. 2: The development of bacterial spot in this test was similar to that in test No. 1 during the first 6 weeks, with the unfertilized plots showing the most infection and defoliation. Incidence decreased as fertilizer rates increased until the 2500-pound rate was attained, and at higher rates of fertilization the plants were relatively free of the disease. After 6 to 8 weeks Cercospora leaf spot developed to such an extent that bacterial spot symptoms were masked. Cercospora leaf spot over-ran all treatments and had almost completely defoliated all plants by the latter part of September.

The two highest rates of fertilization resulted in marginal chlorosis of leaves, stunted plants, and significant reductions in stand.

DISCUSSION

These tests indicate that rates of fertilization affect bacterial leaf spot, do not affect Cercospora leaf spot, and in some extreme cases at high rates of fertilization adversely affect plant growth and survival.

The most outstanding observations were the almost complete absence of bacterial spot on plants fertilized with the equivalent of 5000 pounds of fertilizer per acre, and the consistent increase in disease incidence as fertilization rates decreased. This was conspicuous where

FIGURE 1. Effects of fertilization on development of bacterial spot on pepper: plot fertilized with the equivalent of 5000 pounds per acre of a 12-12-12 fertilizer (left row), and unfertilized plot (right row). Bacterial spot defoliated plants in unfertilized plots causing fruits to be exposed to direct sunlight which later resulted in sunscald.

FIGURE 2. Comparison of bacterial spot damage on representative plants from plot fertilized with the equivalent of 5000 pounds per acre of a 12-12-12 fertilizer (left) and unfertilized plot (right).

unfertilized plots were adjacent to highly fertilized ones (Fig. 1). The factor or factors involved are not known, and the percentages of nitrogen, phosphorus, and potash in the pepper leaves did not furnish a basis for any conclusion.

The fact that Cercospora leaf spot defoliated all plots in test No. 2 is sufficient evidence that rates of fertilization do not affect its development on peppers. This disease causes considerable damage in some years but usually develops late in the season and may be controlled with fungicides when other measures fail.

The mortality, stunting, and leaf chlorosis of pepper plants in plots fertilized with 10,000- and 5000-pound rates of fertilizer in the upland test and in plots fertilized with the former rate in the bottomland test may be attributed largely to the method of fertilizer application. In cases where plants died soon after transplanting, removal of soil revealed that plant roots had been set in the fertilizer zone. The recommended method is to mix fertilizer with soil before setting plants (1), but this was not done in order to preserve some knowledge of fertilizer location. Furthermore, these rates are 8 to 16 times that recommended for pepper (2).

These preliminary tests suggest entirely new avenues of investigation in the control of bacterial spot of pepper. Greenhouse tests are in progress in an effort to reproduce field results, and more comprehensive field tests will be undertaken.

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THE INHIBITORY EFFECTS OF CERTAIN FUNGICIDE FORMULATIONS TO APPLE SCAB CONIDIA

Dwight Powell[1]

Summary

The formulations of dodine (Cyprex), captan, Phaltan and Dyrene checked germination of conidia of apple scab (Venturia inaequalis (Cke.) Wint.) at 2400 ppm for a 21-day period. At lower concentrations, however, a significant spore recovery occurred after 10 days. Of these four materials, dodine exhibited a greater residual toxicity. Dichlone-treated spores revived completely after 10 days. Niacide M did not demonstrate high inhibition initially but its effect was persistent. Zineb was not effective. A captan-zineb mixture gave a fungistatic response equivalent to captan alone. A captan-dodine mixture was slightly synergistic. Conidia did not release normally from the conidiophores when treated with either 50 percent captan or Phaltan 50W at 2400 ppm.

INTRODUCTION

There is a void in our understanding of exactly how a chemical gives protection against a plant pathogen. We know, for example, if we use dodine 65W at 1/2 pound to 100 gallons of water and apply it to apple foliage at intervals of 7 to 10 days through the scab infection period, the leaves of sprayed trees will be free of scab while leaves of adjoining unsprayed trees will become infected. There are many other commercial fungicides that perform in a similar manner. The question, then, is exactly what are the spore-leaf-chemical interactions that produce control? The effect of a chemical on an established infection in the orchard is one of these interactions that is difficult to determine by field observation alone. This experiment was designed to evaluate a number of fungicidal chemicals on the basis of field and laboratory tests using established infections of apple scab (Venturia inaequalis (Cke.) Wint.).

METHODS

Untreated, scab-infected leaves, attached to the tree, were immersed in various concentrations of the test materials. Then, at intervals of 1, 5, 10, and 21 days after treatment these leaves were brought into the laboratory, and spores collected from them were evaluated for percent inhibition.

When the treatments were applied by means of a conventional hydraulic sprayer, spore germination counts were extremely variable within any one treatment. This was probably due to a lack of uniform coverage. This variable was eliminated by immersing rather than spraying the leaves and marking them for future collection. Three leaves were collected from each treatment at each of the collection periods. Two leaf discs, 5 mm in diameter, were removed from each leaf with a standard cork borer. Each disc was taken from the center of a scab lesion. The disc was placed in the center of a cavity of a standard two-celled depression slide (75 x 25 mm) and immersed in 0.1 ml of deionized, sterile water. After 30 seconds, which was sufficient time for spore release, the disc was removed from the water and discarded. The slide was then placed in a moist chamber for 16 hours and spore germination counts made.

The materials and the concentration of each used in the experiment are listed in Table 1. The chemical identity of each formulation is as follows: Captan = N-(trichloromethylthio)-4-cyclohexene-1,2-dicarboximide); dodine = n-dodecylguanidine acetate; Phaltan = a 50% formulation of N-trichloromethylthiophthalimide; Niacide M = manganous dimethyldithiocarbamate, 53.9%, thiram, 10.9%, benzothiazyl disulfide, 2.9%, manganous benzothiazylmercaptide, 2.3%; dichlone = 2,3 dichloro-1, 4-napthoquinone; zineb = zinc ethylene bisdithiocarbamate; Dyrene = 50% 2,4-dichloro-6-(chloroanilino triazine).

RESULTS

In all treatments significant survival occurred from all concentrations except 2400 ppm of

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Table 1. The inhibitory effect of different fungicides on the germination of apple scab conidia 1, 5, 10, and 21 days after treatment.

Treatment	Days after treatment	Percent inhibition at different dilutions (ppm) 2400	1200	600	300	150
Dodine 65W	1	100	100	100	94	80
	5	100	98	98	73	39
	10	100	100	93	80	-
	21	100	95	64	50	-
Captan 50W	1	100	100	100	87	52
	5	100	100	100	42	12
	10	100	93	4	0	0
	21	98	85	-	-	-
Phaltan 50W	1	100	100	100	80	50
	5	100	100	100	31	0
	10	100	84	40	0	0
	21	100	-	-	-	-
Niacide M	1	81	65	72	-	-
	5	81	59	34	-	-
	10	69	59	20	-	-
Dichlone 50W	1		100	78	58	-
	5		60	40	0	
	10		0	0	0	
Zineb 65W	1	0	0	0	0	
	5	0	0			
	10	0	0			
Dyrene 50W	1	100	100	100		
	5	100	100	78		
	10	97	88	66		
	21	94	74			

Table 2. The inhibitory effect of mixtures of captan-zineb and captan-dodine on the germination of apple scab conidia 1, 5, 10, and 21 days after treatment.

Treatment	Days after treatment	Percent inhibition at different dilutions			
Captan 50W plus		1200 ppm	600 ppm	300 ppm	150 ppm
Zineb 65W		1200 ppm	600 ppm	300 ppm	150 ppm
	1	100	100	83	74
	5	100	100	25	0
	10	57	14	-	-
	21	29	-	-	-
Captan 50W plus		1200 ppm	600 ppm	300 ppm	
Dodine 65W		600 ppm	300 ppm	150 ppm	
	1	100	100	100	
	5	98	84	30	
	10	92	-	57	

dodine, captan, Dyrene, and Phaltan. (Table 1). There was little difference in the fungistatic effect of these four materials at the lower concentrations except that dodine showed an effect over a longer period of time. Dichlone-treated spores were 100 percent inhibited for 1 day, but recovered completely after 10 days. In this test dichlone had no fungicidal value and was a good fungistat for only 1 day.

Zineb exhibited no eradicative effects, but Niacide M was partially effective. It is possible that the combination of the various components of the Niacide M mixture provided some eradicative properties.

When spores were collected from the leaf discs, the untreated lesions yielded a concentration of approximately 5 x 10^3 spores per ml. Both captan and Phaltan, at different concentrations, showed an inverse effect on spore release. At low concentrations spore release was normal, but as the concentration increased the spore yield decreased. None of the other chemicals affected spore release.

DISCUSSION

A chemical used for plant protection against fungus diseases can perform either as a protectant, an eradicant, or both. These terms describe the type of response that a chemical may give, but they do not evaluate the quality of that response. A good protectant may be either fungistatic or fungicidal in action. An eradicant normally provides fungicidal action and gives complete kill, but a good fungistat probably could net the same results so far as disease control is concerned.

In this experiment, our so-called fungicides actually performed essentially as fungistats. It is possible that dodine, captan, Phaltan, and Dyrene destroyed the scab pathogen at 2400 ppm but it is also possible that these materials were only active as fungistats at this high concentration. Residue analyses were not included in this study, but it is assumed that where a progressive decrease in inhibition of spore germination occurred, it was congruous with the gradual decline in residual load.

The ineffectiveness of zineb as a contact fungicide is not surprising. While the mechanism of fungicidal action of zineb is not well understood, several theories have been advanced. If its toxicity is dependent upon its chelating properties, then it would not be apt to affect a spore which was not metabolically active. If its fungitoxicity depends on the effectiveness of several breakdown products, then it probably would yield a negative response in this kind of test. In past experiments zineb has been fairly effective in controlling scab. Thus, it must produce a mechanism of fungitoxic action different from that shown by the other compounds used in this experiment.

In observing the performance of captan and Phaltan it is obvious that they had an effect in preventing release of the conidia from the conidiophores. This is a kind of response not normally considered in studying fungicidal action.

Many chemicals are fungicidally active in the laboratory, but can not be effectively formulated for field use. In this test, the results with Niacide M were extremely erratic, with considerable variation between replicates. This indicated a possible weakness in their formulation.

A field-laboratory experiment of this kind is very useful. It exposes fungicides to the rigors of temperature, light, moisture, and air movements which would be impossible to duplicate either in the laboratory or in the greenhouse. Without the right weather conditions, it is of little use. Only light traces of rain occurred from June 12 to July 23 during the time of the test, which gave little interference. After 0.51 inches of rain on July 23, however, the lesions lacked sufficient spores for a suitable spore count and the test was discontinued.

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BRUISING OF APPLES BY STRONG WINDS

L. O. Weaver

In September 1959 apple growers in Maryland made inquiries about the cause of bruise-like spots on certain varieties of apples. The Red Delicious variety appeared most susceptible, but Golden Delicious, Stayman and Cortland were similarly affected. The spots detracted from the appearance of the apples and reduced their market value. Because of the defect about 5 percent of Red Delicious from one orchard failed to grade U.S. No. 1 or better.

The defects appeared as flat-bottomed depressions 1/4 to 3/8 inch in diameter (Fig. 1). More fruits were affected on the pale side than on the blush side. On the pale side of the fruits (next to the branch) the surface color of the spots was a darker green than normal, but on the blush side the affected area was usually a darker red than normal. The spots were subtended by dry brown cells to a depth of 1/8 to 1/4 inch. Some fruits showed only a single spot, but often 10 to 20 spots were found on one side of an apple. In the majority of cases the cuticle was not broken and rot was never observed initiating at the affected area.

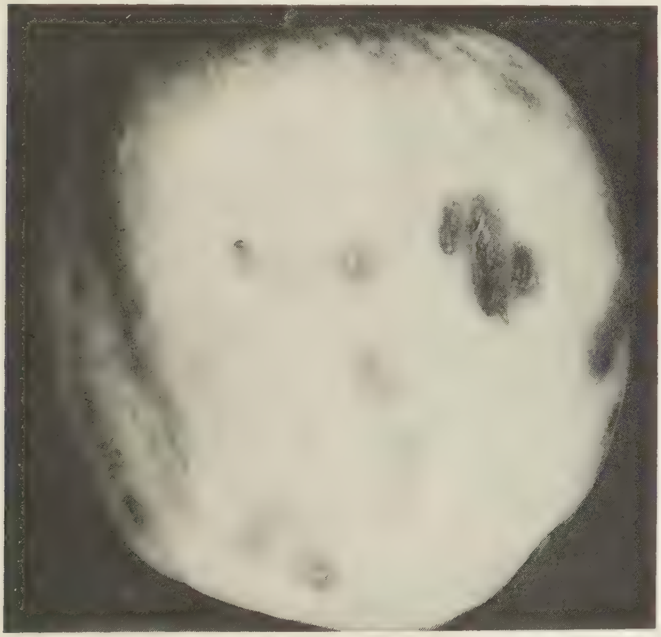

FIGURE 1. Bruising injury on apple resulting from strong winds.

The defect is believed to be bruising injury caused by strong winds during the period June 14-19, 1959 when apples were about 1/4 to 1/2 grown. The position of the spotted apples on the trees would indicate that the bruising was caused by bumping of the fruits against a branch or spur. Considerably more spotted fruits could be found on the inside of the trees on low hanging limbs. Affected fruits were rarely observed by one walking down the outside of the tree row.

A similar spotting of Red Delicious apples was observed in September 1955 in an orchard on the University of Maryland Plant Research Farm. The spotting was considered the result of bruising injury caused by hurricane winds in August of that year.

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EFFECT OF EXOCORTIS DISEASE ON FOUR CITRUS ROOTSTOCKS

J. B. Sinclair and R. T. Brown[1]

Summary

The effects of exocortis on the growth (trunk circumference), yields and symptom expression of Washington navel on Cleopatra mandarin (Citrus reticulata), sweet orange (Citrus sinensis), trifoliate orange (Poncirus trifoliata) and Rangpur lime (Citrus sp.) for a 5-year period are summarized. That Cleopatra mandarin and sweet orange are adversely affected by the virus even though they exhibit no typical exocortis symptoms is indicated by the significantly (.01 level) slower growth in trunk circumferences of infected trees as compared with healthy trees. Scions on rootstocks of trifoliate orange, Rangpur lime and sweet orange showed a significant (.01 level) reduction in yields of diseased trees below those of healthy trees, but there was no significant reduction in yields between healthy and diseased scions on Cleopatra mandarin rootstock. There was a tendency for an increase in the differences between the trunk circumferences of healthy and diseased trees on all four rootstocks, with the difference becoming wider each year.

INTRODUCTION

The exocortis disease of trifoliate orange (Poncirus trifoliata) has been observed in the citrus-growing area of Louisiana for many years. It has been reported on certain trifoliate orange hybrids in California and in other areas where these rootstocks have been used (1). Some Louisiana growers have rogued out large portions of their groves because of this disease. The disease develops slowly on trifoliate orange with traces of bark scaling appearing on roots about 2 years after budding and noticeable dwarfing of the scion (4). The scaling first appears on the side of the larger roots near the soil line and about 4 to 6 years later on the entire rootstock extending to the bud union.

In scaling or shelling, the outer bark separates from the inner bark and peels off in narrow strips 1/2 to 2/3 cm wide and 1/16 to 1/3 cm in thickness. Infected trees are unthrifty, dwarfed, and yields begin to fall below those of healthy trees when the bark scaling appears. Yields of Eureka lemon (Citrus limon) were shown to be reduced 25 to 50 percent 2 years after being budded on rootstocks carrying this disease (3). In 1949 Benton, et al. (1, 2) showed that the disease was caused by a virus.

This paper presents statistical data of the detrimental effects of exocortis on certain rootstocks under Louisiana conditions.

METHODS AND MATERIALS

Seed of four rootstocks furnished by Dr. Frank E. Gardner[2] and Dr. L. C. Knorr[3] were planted in 1950 for a project to be started in 1951 at the Plaquemines Parish Experiment Station to determine if the disease of trifoliate orange was the same as that reported by Benton, et al. (1) and to test the effect of the alleged exocortis virus on four rootstocks as expressed on scions of Washington navel orange (Citrus sinensis). The four rootstocks used were sweet orange (C. sinensis), Cleopatra mandarin (C. reticulata), Rangpur lime (Citrus sp.), and P. trifoliata orange. These were budded in August 1951 with healthy and diseased Washington navel scions. The budwood was taken from three healthy and three diseased trees which were at least 20 years old, growing on trifoliate rootstocks. The trees used as the source of diseased budwood were showing symptoms of the exocortis disease. The trees used as the source of healthy budwood showed no comparable symptoms. The budded trees were placed in a replicated field plot in December of the same year. There were three replications with three healthy and three diseased trees per replication. Yield data and trunk circumferences were recorded in 1955, 1956, 1957, 1958 and 1959 (Tables 1 and 2). A portion of these data has been published else-

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where (13). All Washington navel scions budded on trifoliate orange rootstocks made about 30 cm of growth the first year and died for some unexplained reason. These were successfully rebudded the following year, thus making the Washington navel scions on these rootstocks a year younger than the others.

Table 1. Treatment means for total yields in pounds of fruit per tree of Washington navel orange scions on four exocortis-infected or healthy rootstocks for the year of harvest indicated.

Rootstock	Treatment	Year of harvest: 1955	1956	1957	1958	1959
Cleopatra mandarin	Healthy	7.3	7.7	114.8	193.8	131
	Exocortis	20.3	12.6	113.6	187.0	138
Sweet orange	Healthy	20.4	11.2	118.5	188.1	172
	Exocortis	17.4	12.3	95.4	157.9	156
Trifoliate orange	Healthy	0.0	0.0	32.8	108.0	138
	Exocortis	0.0	0.0	9.7	31.8	32
Rangpur lime	Healthy	10.2	3.1	155.2	210.5	168
	Exocortis	15.3	6.9	90.0	113.5	85
LSD .05		ns	ns	12.0	21.3	20
LSD .01		ns	ns	16.7	30.6	28

Table 2. Treatment means for trunk circumferences in cm of Washington navel orange scions on four exocortis infected or healthy rootstocks for the number of years after budding indicated.

Rootstock	Treatment	Years after budding: 4	5	6	7	8
Cleopatra mandarin	Healthy	24.3	33.7	35.9	40.5	43.5
	Exocortis	23.3	30.3	31.8	35.2	38.1
Sweet orange	Healthy	28.0	35.1	40.0	43.8	45.3
	Exocortis	23.8	29.5	33.7	39.4	41.6
Trifoliate orange	Healthy	12.8	16.6	23.1	27.4	---[a]
	Exocortis	10.0	10.7	14.7	15.0	---
Rangpur lime	Healthy	25.4	32.5	35.3	39.4	41.3
	Exocortis	20.6	23.9	26.2	28.8	31.0
LSD .05		1.3	1.8	1.9	1.9	1.9
LSD .01		1.8	2.4	2.6	2.6	2.7

[a]No data, see text.

EXPERIMENTAL RESULTS

In August 1953, about 2 years after the original budding, small gum pustules formed on the main roots and trunk near the soil line or higher, but never about the bud union, of Rangpur lime rootstocks that had been budded with diseased Washington navel scions. Gum exuded from the pustules, and the outer bark began to shell-off. The leaves showed some chlorosis in June preceding the appearance of the gumming. Off-season flowering and the rate of growth was retarded. The symptoms were similar to those reported for Rangpur lime disease by Olson (8), which led Brown (4) in 1954 to believe that the two diseases might be caused by the same virus. In addition to this test, a rootstock experiment using scions of Valencia orange and Ruby Red grapefruit (*Citrus paradisi*) carrying exocortis showed that trees budded on Rangpur lime had

similar gumming and bark shelling. Where a healthy source of budwood was used no such symptoms have been observed (4). Findings by Moreira (6, 7) in 1955, Olson (9), and Olson and Shull (10) in 1956 and Reitz and Knorr (12) and McClean (5) in 1957 added further evidence to Brown's conclusion that the Rangpur lime disease and exocortis disease are the same or are closely associated (4).

The yield data were analyzed and presented for each year of harvest in a way to reduce as much as possible the influence of weather conditions and other factors which might affect yields from one year to the next (Table 1). Analysis of the yield data for the 5 years showed no significant differences between the means of total yields from healthy and diseased trees in 1955 and 1956, although there was a tendency for trees on diseased scions to have yields above those on healthy scions. Washington navel scions on healthy rootstocks gave yields in 1957 and 1958 significantly (.01 level) above those on diseased rootstocks, and those on Cleopatra mandarin with diseased rootstocks gave yields below those of healthy rootstocks, but the difference was not significant. Yield differences between the two treatments of all four rootstocks were wider in 1958 than 1957, with the widest difference appearing between healthy and diseased scions on Rangpur lime rootstocks and the least difference between healthy and diseased scions on Cleopatra mandarin. Yields tended to be lower in 1959 than 1958. There was no significant difference between healthy and diseased scions either on Cleopatra mandarin or sweet orange. The differences between yields from healthy and diseased scions on trifoliate orange and Rangpur lime continued to be significant (.01 level).

Another, and perhaps more reliable, criterion for measuring the effects of this disease is the comparison of trunk circumferences of healthy and diseased trees taken 15 cm above the bud union. These data were taken for each of the 5 years, recorded in cm and statistically analyzed (Table 2). The data are presented in years after budding, so that the scions on trifoliate orange rootstocks could be compared with the other scions of the same age.

Trunk measurements for the 5 years showed that the circumferences of healthy trees on sweet orange and Rangpur lime rootstocks were significantly higher (.01 level) than those of diseased trees on these stocks. There was no significant difference between the trunk circumferences of scions on healthy and diseased Cleopatra mandarin rootstocks 4 years after budding, but highly significant differences between the two treatments were found in the 4 succeeding years. In each year the greatest difference was found between the diseased and healthy scions of Rangpur lime; the least difference between diseased and healthy scion of Cleopatra mandarin, except in the seventh year after budding, when the least difference was shown to be between the scions on healthy and diseased sweet orange rootstocks. There was a tendency for an increase in the difference between treatments on all four rootstocks, with the difference becoming wider each year. Sweet orange was the exception, where the difference between treatments was 2.9 cm less in the seventh year than in the sixth year.

DISCUSSION

The wide differences in trunk circumferences and yields between Washington navel scions on diseased and healthy rootstocks of trifoliate orange and Rangpur lime would be expected since both these rootstocks exhibited definite exocortis symptoms. Sweet orange and Cleopatra mandarin rootstocks, on the other hand, are carriers of the disease even though they have not shown any definite exocortis scaling. Yet, the differences in trunk circumferences between virus-free and virus-infected Washington navel scions on these tolerant rootstocks indicate that exocortis has a deleterious influence even on these rootstocks. Yield data indicate sweet orange is affected more than Cleopatra mandarin by the exocortis virus.

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DEPARTMENT OF PLANT PATHOLOGY AND PLAQUEMINES PARISH EXPERIMENT STATION, LOUISIANA AGRICULTURAL EXPERIMENT STATION, BATON ROUGE

MELILOTUS ITALICA, A NEW HOST FOR UROMYCES STRIATUS[1]

E. E. Leppik[2]

Abstract

Reported is the first rust case on Melilotus italica, the first known host for Uromyces striatus Schroet. from the genus Melilotus. Both uredio- and teliospores (hitherto rarely found in America) were collected in Ames, Iowa in the fall of 1959. This rust belongs to the variety Uromyces striatus medicaginis (Pass.) Arth., hitherto observed only on Medicago. Slight morphologic differences of this variety and some variation in its biologic specialization indicate that a new microcyclic form of this rust is developing on the American continent with a host range of its own.

Melilotus italica, Italian sweetclover, has been repeatedly introduced into the United States from various Mediterranean countries. So far no rust has been observed on this plant, either in the United States or in the native Mediterranean area (1, 2, 3, 4, 5). According to Guyot (4), Uromyces anthyllidis (Grev.) Schroet. and U. baeumlerianus Bubak infect various species of Melilotus in Europe, except M. italica. Yet none of these rusts are reported from America (1, 2, 4).

The rust on Melilotus italica was first observed in August 1959 on plants derived from seed introduced from Spain (PI 226538, 244287) and grown at the North Central Regional Plant Introduction Station, Ames, Iowa. Urediopustules frequently developed on leaves, mixed with a few telia in late fall (Fig. 1). Since this was the first rust observed on this host plant, the question arose whether it might have been imported by seed sample. Later observations and the identification of the fungus showed that this was not the case.

FIGURE 1. Rust pustules of Uromyces striatus medicaginis (Pass.) Arth. on leaves of Melilotus italica.

Our rust appeared identical with Uromyces striatus medicaginis (Pass.) Arth., which is common on Medicago spp. in America and occurred frequently on alfalfa at Ames, occasionally in the same field with Melilotus italica. In our collection urediospores were globoid 16-20 μ by 18-22μ with three or four equatorial pores. Teliospores were globoid or ellipsoid, 16-20μ by 20-24μ, strongly striate with elongate warts.

In Europe Uromyces striatus is a heteroecious rust on Euphorbia (0, I) and Medicago (II, III). In America mainly urediospores develop, which infect the same host plant. Telio-

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[1] Disease Reports of the NC-7 Regional Plant Introduction Station, United States Department of Agriculture, Ames, Iowa. No. 3.

[2] Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture. The author is indebted to W. H. Skrdla for assistance in facilitating this study.

spores are rarely found in America, except for the above-mentioned case on Melilotus italica. No pycnia or aecia have ever been found in America.

In the New World this rust overwinters in the tissue of perennial Medicago, or by means of urediospores in southern States. Melilotus italica is an annual and must be infected every season from some alfalfa field. Under these circumstances, full evidence exists that the new rust on Melilotus italica is an American variety of Uromyces striatus and not a foreign disease.

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AN APPARENTLY NEW ROOT NECROSIS DISEASE OF RHUBARB[1]

Harry H. Murakishi

Summary

A root necrosis disease of rhubarb was found to be caused by an unidentified, non-sporulating fungus. Symptoms reproduced in the greenhouse consisted of chlorosis and wilting of the lower leaves and necrotic streaks in the vascular tissue 3 to 6 weeks following inoculation.

A previously unrecorded root necrosis affecting rhubarb (Rheum rhaponticum L.) was found on a farm in southeastern Michigan in 1956. Subsequently it has been observed in three other fields in the area. The symptoms of the root disorder resembled Verticillium wilt (9, 11, 12, 13), but attempts to isolate this fungus or any other soil-borne pathogen previously reported on rhubarb (1, 2, 3, 4, 5, 6, 7, 8, 10) were unsuccessful. A study was therefore made to determine the nature and cause of the apparently new disease of rhubarb.

FIELD SYMPTOMS

Aboveground symptoms manifested in the field consisted of yellowing around the margins of lower leaves and a somewhat stunted appearance of the plants. When these plants were dug from the soil and examined all were found to be affected with necrotic streaks of the vascular tissue in the main and lateral roots (Fig. 1). Plants with no obvious aboveground symptoms frequently had light to moderate root necrosis also. In severely affected plants the necrotic streaks extended into the crown. The necrotic streaks were hard in texture compared with the softer, healthy tissue and could be easily removed from the roots by means of scalpel and forceps.

ISOLATION

Roots were washed free of soil and dipped in 70 percent alcohol and then slit open with the aid of a flamed scalpel. A small portion of the necrotic tissue was aseptically removed and plated on water or potato-dextrose agar. Although Fusarium, Penicillium, Aspergillus, and a bacterium were occasionally encountered, the organism most consistently isolated was a slow-growing, dark, grayish fungus with fine, septate mycelium. It was also isolated from petioles of diseased plants. The fungus has so far failed to sporulate in culture on a number of laboratory media including straw agar on which Wilhelm, et al. (12) were able to obtain microsclerotia of Verticillium albo-atrum isolated from diseased rhubarb. Neither did the fungus sporulate on aseptically removed necrotic root tissue or petioles placed in a moist chamber, a method found by McKay (9) to induce sporulation in 3 days of V. albo-atrum, the cause of a rhubarb root disease in Ireland.

PATHOGENICITY

Three methods of inoculation were tested. Roots of healthy seedlings were dipped in a homogenized, mycelial suspension of the fungus; surface-sterilized seeds were planted in a mixture of steamed soil and corn meal-sand cultures of the fungus; and seeds were coated with a homogenized mycelial suspension before planting. All methods were successful in inducing root necrosis symptoms, but the root-dip method of inoculation gave the most consistent infection. Following this procedure, the pathogenicity of seven isolates of the fungus grown in potato-dextrose broth shake cultures were individually tested on 4-month-old Victoria rhubarb seedlings grown in 6-inch pots of either steamed soil or sphagnum moss. The latter medium was used so that roots could be easily removed and examined periodically for symptoms. Roots

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FIGURE 1. Necrotic streaks of vascular tissue induced by experimental inoculation. Healthy, uninoculated root on extreme left.

FIGURE 2. Foliar symptoms 6 weeks following root inoculation. Healthy, uninoculated plants on left. Inoculated plants on right.

of control plants were dipped in sterile, potato-dextrose broth before planting. Three weeks after inoculation, lower leaves became chlorotic at the margins and wilted (Fig. 2). Root symptoms similar to those seen in the field were found 3 to 6 weeks after inoculation. Six of the seven isolates were found to be pathogenic on rhubarb. Together, the six pathogenic isolates induced root necrosis in 65 out of 78 inoculated plants. In the uninoculated control, consisting of 18 plants, all grew normally and were without symptoms.

A limited host range test of the causal fungus of rhubarb root necrosis was made. Plants

selected were representative of the vegetable crops grown in the area. Two isolates previously found to be pathogenic on rhubarb were used to inoculate seedlings. Six to 10 plants each of the following hosts were inoculated by the root-dip method: eggplant (var. Black Beauty), tomato (Rutgers and Fireball), lettuce (Great Lakes), spinach (Long standing Savoy), pepper (Allbig), common buckwheat, and rhubarb (Victoria). After 8 weeks all plants were examined for foliar and root symptoms. None except rhubarb were affected.

DISCUSSION

Although similar in symptomatology to Verticillium, the causal fungus of rhubarb root necrosis failed to sporulate in culture or to infect hosts such as tomato, eggplant and pepper, which is contrary to the reported behavior of Verticillium (11). The sterile fungus Rhizoctonia has been reported as the cause of a foot rot of rhubarb. Clinton (2) found that Rhizoctonia caused lesions and necrosis of the petioles of rhubarb while Gleisberg (4) observed that the fungus killed the plants. In the case of the root necrosis disease, no necrosis of the petioles were ever seen in the field. Furthermore, the fungus in question does not have the type of mycelial branching characteristic of Rhizoctonia.

In southeastern Michigan, rhubarb is grown almost entirely for forcing during winter. Crowns and roots are started from vegetative cuttings or divisions which are grown for 2 to 3 years in the field before they reach sufficient size for forcing. Conversations with growers indicated that the original crowns from which divisions were made ranged in age from 20 to 42 years. The prevalence of the disorder on the four farms is probably due in large measure to the method of propagation. Careful selection of planting material should aid in reducing the severity and incidence of the disorder. Studies are in progress to determine the identity of the causal fungus and its eventual effect on yield and quality of forced rhubarb.

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, MICHIGAN STATE UNIVERSITY, EAST LANSING

REDUCTION IN YIELD OF COTTON CAUSED BY DISEASES IN 1959

Compiled by the Cotton Disease Council, Committee on Disease Losses: Harlan E. Smith, Chairman; A. L. Smith, W. E. Cooper, Leonard Lett.

The accompanying tabulated summary of the 1959 Cotton Disease Loss Estimates is the eighth report submitted by the committee on cotton disease losses. The summary has been compiled from 56 State estimates. The accuracy of the estimates is highly reliable because most of the same cooperators have contributed for the last 8 years and, as a consequence, their methods and techniques are becoming uniform. The committee feels that considerable credence can be placed in the report.

Table 1. ESTIMATED REDUCTION IN 1959 COTTON YIELD AS A RESULT OF DISEASE DAMAGE.

Disease	Calif.	Ariz.	N. Mex.	Tex.	Okla.	Mo.	Ark.	La.	Miss.	Ala.	Ga.	S.C.	N.C.	Tenn.	Bales Lost	Percent Loss
1. FUSARIUM WILT *Fusarium vasinfectum*	--	--	--	0.36	0.40	4.00	1.26	2.50	2.30	1.00	2.25	0.88	1.75	0.25	158,583	0.93
2. VERTICILLIUM WILT *Verticillium albo-atrum*	1.33	2.95	6.12	1.85	0.93	3.50	3.30	0.20	0.70	tr	--	--	0.40	1.75	287,536	1.69
3. BACTERIAL BLIGHT *Xanthomonas malvacearum*	0.66	tr	0.46	5.91	3.83	3.50	1.00	0.20	0.25	0.40	1.25	1.37	0.75	0.30	419,119	2.48
4. ROOT ROT *Phymatotrichum omnivorum*	0.66	2.29	tr	4.43	0.11	--	0.03	tr	--	--	--	--	--	--	272,681	1.61
5. ANTHRACNOSE BOLL ROT *Glomerella gossypii*	--	--	tr	tr	0.66	0.50	0.03	--	2.00	2.00	0.70	1.87	1.00	0.50	79,962	0.47
6. SEEDLING DISEASES *Rhizoctonia*, etc.	2.20	1.86	2.26	1.75	3.50	1.00	1.36	2.50	2.25	1.50	5.50	1.85	2.50	2.00	343,809	2.09
7. ASCOCHYTA BLIGHT *Ascochyta gossypii*	--	--	--	0.38	--	0.20	0.03	--	tr	tr	0.50	0.20	0.10	0.10	27,483	0.16
8. BOLL ROTS *Rhizopus*, etc.	3.50	1.32	0.37	0.46	1.33	0.10	4.06	3.00	4.50	3.00	1.25	8.50	2.75	1.00	380,109	2.24
9. ROOT KNOT *Meloidogyne* sp.	1.33	1.46	1.30	0.50	0.16	4.50	0.10	0.10	0.45	3.00	3.00	4.75	2.50	0.50	193,050	1.13
10. OTHERS	0.66[a]	0.95[b]	0.73	0.70	--	5.00	0.14	tr	--	--	0.75	0.75	0.30	0.10	107,778	0.63
TOTAL PERCENT LOSS	10.34	10.83	11.24	16.34	10.92	22.30	11.31	8.50	12.45	10.90	15.20	20.17	12.05	6.50		13.43
TOTAL BALES LOST	221,423	90,090	41,409	878,914	47,195	144,935	198,298	45,983	223,260	88,080	94,103	104,854	44,528	46,229	2,270,301	
YIELD IN THOUSAND BALES 1959[c]	1,920	750	327	4,500	385	505	1,555	495	1,570	720	525	415	325	665		

[a] Leaf crumple virus [b] Crazy top and rust (*P. cacabata*) [c] Dec. 1, 1959 USDA AMS Estimate

COTTON DISEASE COUNCIL, COMMITTEE ON DISEASE LOSSES

CERCOSPORA LEAF SPOT OF TUNG IN MISSISSIPPI[1]

Douglas C. Bain

Abstract

Cercospora leaf spot (Mycosphaerella aleuritidis (Miyake) Ou) of the tung oil tree (Aleurites fordii Hemsl.) is assuming serious proportions in south Mississippi. Symptoms of the disease on stems, twigs, nuts, petioles, and leaves are described. Overwintering of the fungus north of the Tung Belt is noted. Results of inoculation tests indicate a difference in reaction of seedlings which suggests the possibility of selecting for resistance. Control measures are discussed.

The writer's attention was called to the severity of Cercospora leaf spot (Mycosphaerella aleuritides (Miyake) Ou = Cercospora aleuritidis Miyake) of the tung oil tree (Aleurites fordii Hemsl.) in 1955 by Dr. G. F. Potter of the U.S.D.A. Tung Station at Bogalusa, Louisiana. According to Potter, the disease was assuming serious proportions in the Tung Belt and was causing premature defoliation in some instances. Concern was based mainly on the fact that oil is being synthesized during August and early September and loss of functional leaf area could adversely affect oil content of the nuts. In the consideration of control measures, knowledge of certain aspects of the disease and life history of the causal organism is desirable. Limited investigations revealed hitherto unreported information which should be of value to those interested in diseases of tung.

THE DISEASE

Ou (2) ably described the disease and demonstrated the relationship between the conidial and perfect stages as they occur in the Chungking area of China. There are, however, differences between the disease and fungus as they are described in China and as they occur in Mississippi. Leaf spotting becomes evident usually in the latter part of May in south Mississippi; the time varies with the temperature. Frequently symptoms appear on leaves of seedlings scattered throughout orchards before they are seen on larger trees. Spots are reddish brown, variable in size, somewhat angular to circular, and may be few or numerous per leaf (Fig. 1). In some instances heavily spotted leaves present a ragged appearance. Diseased leaves are often abscissed prematurely. Not described by Ou (2) are the dark-brown to black, ovate-elliptic, slightly sunken lesions which occur on petioles, young twigs, and stems of seedlings (Fig. 2). These lesions may be up to 2 cm or more in length and 1.5 cm in width. Fruiting structures and conidia have also been observed in dark, circular, sunken lesions on hulls of mature nuts. An abundance of conidia is produced on the lower surface of diseased leaves but rather sparsely on twigs, stems, petioles, and nuts.

FIGURE 1 (left). Symptoms of the early stages of Cercospora leaf spot on the lower surface of tung leaf.

FIGURE 2 (right). Cercospora lesion on stem of tung seedling.

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THE FUNGUS

Stromata, on which tufts of conidiophores are borne, are easily seen with the aid of a hand lens. Conceivably, they could be mistaken for minute pycnidia. In general, the conidia are hyaline although they may become slightly olivaceous, 3-12 septate, and 63-110μ x 3.5-4 μ. These measurements are in the range of those described by Miyake (1) and Ou (2). Conidia were produced in abundance on diseased leaves that had overwintered outside at State College. Just when perithecia become evident in south Mississippi is not definitely known, but maturing perithecia were evident in old lesions by April 1 on diseased leaves brought from Poplarville to State College (about 230 miles north of Poplarville, Mississippi) and placed in a wire basket outside the previous November. Ascospores average 10.8 x 3.6μ, slightly wider than those described by Ou (2). Perithecia have not been observed on petiole, twig, stem, or nut lesions. It is considered significant that overwintering of the fungus and production of the perfect stage was accomplished about 150 miles north of the Tung Belt. At higher temperatures farther south, diseased twigs, stems, debris of diseased leaves and nut hulls can easily supply sources of inoculum not long after leaves emerge in the spring.

INOCULATION EXPERIMENTS

Infection of leaves and stems was obtained in a number of inoculation experiments in the greenhouse at State College in January and February of 1956. Isolates from leaf and stem lesions were used on seedlings of the variety Isobel[2]. Leaves were sprayed with mycelial suspensions but stems were inoculated by inserting the fungus under slits in the bark. Leaf infection became apparent in 25 to 32 days but evidence of stem infection was obvious in 15 days. Although isolates from leaves produced stem lesions, they were not so large as those caused by isolates from stems. Young leaf lesions are very small, angular, and dark red with a narrow pale green halo. In the first series of inoculation tests there were marked differences in reactions of various seedlings; lesions on some plants were only "flecks" and remained so, while on other plants lesions increased considerably in size. Results of later inoculation experiments of "budded" plants[2] showed that lesion development was uniform. In this connection, certain varieties of tung trees in variety test plots at Poplarville Tung Station were almost completely free of leaf spot while other varieties nearby were heavily infected in 1955 and 1956. On the basis of results of inoculation experiments, as well as field observations, it would seem there is the possibility of selecting for resistance.

DISCUSSION

In a consideration of control measures, occurrence of the fungus -- which is easily capable of overwintering on the various plant parts -- must be taken into account. Large piles of nut-hull debris are left scattered about orchards by hulling machines. Dead leaves and nuts on the ground under trees are not easily reached by machinery. These, as well as infected twigs and stems, could supply abundant inoculum for infection in the spring. Conidia produced from primary infections add to the inoculum potential. Little, if any, control could be gained by sanitary practices or by attempting to plow under debris. There is reason to believe that spraying with any of the fixed-copper compounds or with Bordeaux mixture would hold the disease in check if applications were started early enough in the spring and continued at intervals until late in the growing season. Spraying, however, does not appear to be economically feasible. Since there apparently is a considerable difference in seedling and varietal reaction, selection for resistance and development of commercially desirable resistant varieties would seem to offer the best means of control.

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[2] Supplied through the courtesy of Dr. G. F. Potter.

SQUIRRELS AS POSSIBLE VECTORS OF THE OAK WILT FUNGUS IN IOWA[1]

Arthur W. Engelhard[2] and W. H. Bragonier[3]

Abstract

Feeding habits of squirrels on fungus mats and pads on oaks killed by Ceratocystis fagacearum (Bretz) Hunt and on healthy trees of the red and white oak groups were observed in Iowa during 1952, 1953 and 1954.

Extensive squirrel feeding on mats and pads occurred in the spring and fall in Pilot Knob State Park in northern Iowa. The spring feeding coincided with the period when 1) squirrels fed extensively on the buds, shoots, leaves, flowers and bark of healthy oak trees; 2) squirrels cut off leaves and twigs to be used in building nests; and 3) natural spread of the oak wilt fungus occurs.

On May 6, 1955 a gray squirrel (Sciurus carolinensis Gmelin) was observed feeding on a perithecium-bearing fungus mat and pad on a wilt-killed red oak (Quercus rubra). The squirrel was shot. Cultures made from both the mouth and stomach contents produced the oak wilt fungus.

Observations made in Pilot Knob, Dolliver and Pike's Peak State Parks showed that a higher percentage of the mats and pads were eaten by squirrels in areas where fruiting was abundant than in areas where it was sparse.

Circumstantial evidence strongly indicates that in some areas squirrels may be closely associated with the spread of the oak wilt fungus, especially where the disease is well established and fungus mats and pads are abundant. This association may be either direct, by transmitting the pathogen, or indirect, by making wounds on healthy oak trees to which inoculum-laden potential vectors, such as insects, might be attracted.

INTRODUCTION

The diet of squirrels is varied and differs from one time of year to another. It consists largely of parts of trees, such as buds, young shoots, bark, flowers, leaves, acorns and nuts, and of fungi. Squirrels feed on hard woods (2, 3, 4, 5, 12, 14, 16, 20), fruit trees, (3, 20), and conifers (1, 3, 5, 8, 9, 12, 15, 18). They eat and store mushrooms (6, 16), feed on oak wilt fungus mats and pads on wilt-killed oaks (7, 11, 14, 21) and on the blister rust fungus on white pines (17, 26).

In the spring young shoots and buds furnish a large part of the squirrels' diet (5, 12, 14). Leaves and twigs are used extensively for building nests for the two broods produced during the year. Squirrels eat tree bark during the summer (12). They cut and feed upon twigs in the winter when snow covers the ground and other food is not readily available (3, 20). It appears that wherever squirrels are present wounds of various types will be made on trees as a result of their activities.

The importance of wounds on oak trees, especially in the spring months, as infection courts for the oak wilt fungus (Ceratocystis fagacearum (Bretz) Hunt) has been pointed out (10, 19, 22, 23, 25). Evidence indicates that in order for long distance spread of C. fagacearum to take place wounds of some type are necessary on healthy oak trees. Therefore, wounds made by squirrels are potentially important in the spread of the oak wilt fungus.

It was experimentally demonstrated that caged squirrels can transmit the oak wilt fungus to healthy oak seedlings. The transmission occurred for as long as 12 hours after the animals had eaten the fungus inoculum (13).

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This paper discusses the feeding and gnawing habits of squirrels on mat- and pad-bearing wilt-killed or infected oaks and on healthy oak trees. These are activities which might implicate squirrels either as direct vectors of C. fagacearum or as wounding agents facilitating the spread of the pathogen.

FIELD OBSERVATIONS

In northern Iowa mats and pads of the oak wilt fungus are produced in the cambial region of wilt-killed oak trees of the red oak group in the fall, in the spring, or in both fall and spring following death of the host. As the mats and pads grow, cracks appear in the bark over them and a fruity odor is emitted. Insects, rodents and birds are attracted to the fungus structures, possibly because of the odor.

Squirrels readily feed on the fungus mats and pads. They chew or rip off pieces of bark overlying the fungus structures to gain access to them. The area of bark removed usually does not exceed 3 inches in length and 2 inches in width (Fig. 2), but in some instances strips of bark a foot or more in length are taken off. After removing the bark, the squirrels consume the central fungus pad, and often portions of the surrounding mat.

The feeding habits of squirrels on fungus mats and pads on oaks[4] killed during 1952, 1953, and 1954 were observed and recorded (Table 1). The data indicate squirrel feeding on mats

Table 1. Frequency of squirrel feeding on fungus mats and pads on wilt-killed oaks, Pilot Knob and Dolliver State Parks, Iowa.

Year wilted	State Park	Number of oaks with mats and pads	Number of oaks with squirrels feeding on mats and pads
1952	Pilot Knob	31	22
1953	Pilot Knob	145[a]	134[a]
1953	Dolliver	18	1
1954	Pilot Knob	23[b]	21[b]
1954	Dolliver	20	0

[a] Includes one bur oak.
[b] Includes two bur oaks.

and pads was most prevalent where large numbers of mat- and pad-bearing trees were present. In Pilot Knob State Park in north-central Iowa several hundred mat- and pad-bearing oaks were present during each of the three years data were recorded. Squirrel feeding was observed on mats and pads on 22 of 31 trees recorded that wilted in 1952, 134 of 145 in 1953, and 21 of 23 in 1954. In Dolliver State Park the oak wilt centers were widely scattered and averaged less than two mat- and pad-bearing trees per center each year. During a 2-year period, evidence of feeding on the fungus structures by squirrels was observed on only one of 38 mat- and pad-bearing oaks. In this instance feeding had occurred on only one mat and pad on the tree (Table 1). Although no data were recorded, very little feeding was observed on the scattered mat- and pad-bearing oaks in Pike's Peak State Park in 1953. Squirrels were abundant in all three parks.

In Pilot Knob State Park many trees were found on which more than 40 mats and pads had been eaten by squirrels. One northern pin oak (Quercus ellipsoidalis), 12 inches d.b.h. and 40 feet tall, was felled and the bark peeled during the winter and spring of 1953-54. The tree had at least 246 mats and pads (11). There were 108 wounds in the bark where squirrels had gnawed. Eighty-four of the bark wounds were over remnants of mats and pads. The other 24 wounds were on smaller limbs which had deteriorated in the rain and snow, making uncertain the identification of what appeared to be parts of old mats and pads. However, it is quite likely that these additional wounds were over mats and pads, as only two of many similar wounds observed on trees producing fresh mats and pads were not associated with the fungus structures.

Mats and pads were produced most abundantly in Pilot Knob State Park in late August, September, October, early in November, April, and May (11). These were also the months

[4]Mostly northern pin oaks (Quercus ellipsoidalis), red oaks (Quercus rubra) and bur oaks (Quercus macrocarpa).

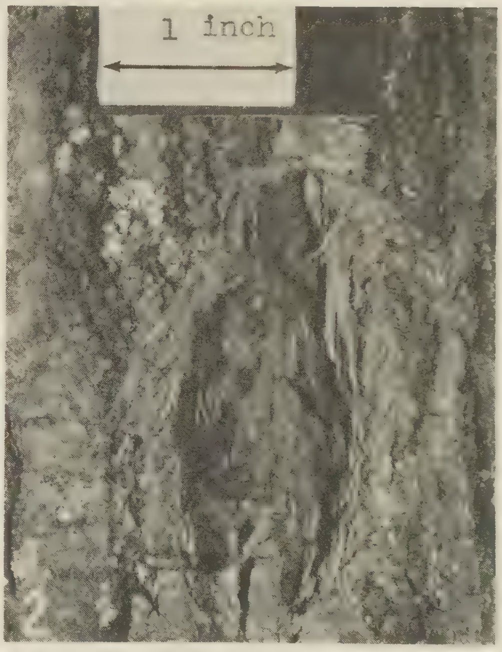

FIGURE 1. A portion of the trunk of a wilted red oak. The squirrel removed the bark over a mat and pad containing perithecia and consumed parts of the fungus structure. Ceratocystis fagacearum was cultured from the mouth and stomach contents of the squirrel.

FIGURE 2. Enlarged view of the hole in the bark shown in Figure 1. Photos by Lou Facto.

during which most mat- and pad-feeding by squirrels occurred. Thus, according to these data on observations, the frequency of mat- and pad-feeding by squirrels was correlated directly with the abundance of the fungus structures present.

LABORATORY CULTURING

On May 6, 1955 a female gray squirrel (Sciurus carolinensis Gmelin) was observed climbing a wilt-killed red oak (Quercus rubra) in Pilot Knob State Park. Quickly she removed the bark overlying a perithecium-bearing mat and pad and consumed a part of the fungus structure (Figs. 1, 2). The squirrel was shot and taken to the laboratory for examination and culture tests. Large, thin-walled cells similar to those comprising the fungus pads of C. fagacearum were found in the stomach. Isolation tests were made from the stomach contents, and also by smearing teeth and mouth parts directly onto agar, and by smearing sterile cotton swabs which had been passed around in the mouth directly onto agar. Thirty-eight Petri dishes containing 2 percent potato-dextrose agar (PDA) containing a small amount of lactic acid were used for these tests. Four of the dishes prepared from smears from the mouth parts and cotton swabs yielded C. fagacearum. Solutions were also prepared from blood taken from the mouth, from contents of the stomach, and from sterile cotton swabs passed around in the mouth. The solutions were serially diluted (24). A total of 203 Petri dishes of water agar or PDA were "seeded"

with the solutions. One PDA plate "seeded" from the original solution prepared from blood taken from the mouth and 1 water agar plate "seeded" from the first dilution prepared from stomach contents yielded the oak wilt fungus.

DISCUSSION

Activities centered about the feeding and gnawing habits of squirrels throw suspicion on these animals as vectors of the oak wilt fungus or as wounding agents facilitating the spread of the oak wilt disease. Squirrels may feed extensively on mats and pads in areas where the fungus structures are present. They also carry the viable fungus in their mouths. They wound healthy trees by cutting off buds, shoots, leaves, catkins, acorns and by tearing or chewing off bark. Experimentally, it has been demonstrated that caged squirrels can transmit the oak wilt fungus to healthy oak seedlings for as long as 12 hours after coming in contact with the inoculum (13). Feeding in nature by squirrels on mats and pads and feeding and gnawing on healthy oaks during the same period of the year strongly implicates these animals as vectors of the oak wilt fungus.

Experimental transmission of the oak wilt fungus by insects has been obtained when inoculum-laden insects were caged so they had easy access to fresh wounds (10, 22, 25). Fresh wounds are very important in the long distance spread of the oak wilt fungus by insects (10, 22, 23). During the spring of 1954 in Pilot Knob State Park oak wilt occurred in three red oaks soon after squirrels stripped large areas of bark from the trees. One could speculate either that squirrels transmitted the pathogen while making the wounds or that insect vectors transmitted the pathogen to the fresh wounds made by squirrels.

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EXPERIMENTAL GREENHOUSE CONTROL OF CROWN GALL AND OLIVE KNOT WITH ANTIBIOTIC DRENCHES

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Abstract

Woody types of crown gall (Agrobacterium tumefaciens) on apricot and peach, and olive knots (Pseudomonas savastanoi) were successfully controlled by antibiotic drenches consisting of streptomycin, terramycin, or a combination of both in an iso-amyl-kerosene-lanoline-vaseline mixture. The drench was applied with a paint brush on intact galls. The galls were killed within 4 to 7 days in a warm greenhouse.

The occurrence of crown gall on almond, apricot, peach, and walnut roots or crowns is common in many commercial orchards, since the crown gall organism is a wound parasite and wounding of plant parts by cultivators and plows is unavoidable. The disease will always have to be dealt with and economical and simple means of eliminating it from affected parts are desirable.

The cutting off of crown galls by surgery, followed by the disinfecting of wounds with mercuric chloride -- or even with some antibiotics, such as streptomycin -- can be effective, but the cost is high and may be prohibitive in some situations. In 1941-42 the senior author experimented with the eradication of crown galls on almond and peach roots using an elgetol-methanol drench (3, 4). The surface of an intact gall was painted, together with a strip (about 1-inch wide) of the healthy bark around the gall. Since that time it was discovered that the idea of painting a whole gall was suggested in California as far back as 1873 (2). It was stated that "for removing knots from plum trees, he takes a paint brush, dips it in spirits of turpentine, and thoroughly saturates the knot, being careful not to touch the tree except in the diseased parts. The turpentine kills the excrescence." The material used by this pioneer was thoroughly tested by the authors and has been found to be extremely injurious. Turpentine has a strong affinity for plant tissues and is rapidly translocated within the plant, especially at high temperatures.

With the advent of antibiotics, tests were conducted on the effects of well-known antibiotics on plant pathogenic bacteria both in vitro and in vivo (1, 5, 6, 7, 8, 9, and 10). Penicillin, streptomycin, and terramycin gave good results in vitro. Because streptomycin appeared to be the most stable of the known antibiotics, more work has been done with it than with other antibiotics. Brown's (7) experiments on the killing of Xanthomonas pruni in grafting wood of peach with streptomycin by translocation from a solution, and the demonstration by Mitchell et al. that streptomycin can penetrate into the plant from streptomycin lanolin paste, stimulated plant pathologists to try similar methods for the control of artificially induced crown galls (7, 8). The work carried on at this station indicated that crown gall tissues are difficult to penetrate if aqueous solutions of various chemicals including antibiotics are used. Crown galls can be held in check if the gall is first cut off from the plant and the wound then painted with streptomycin solutions or pastes containing from 500 to 1000 ppm of the active antibiotic or 200 ppm terramycin. It was the aim of this work to develop a formulation applicable to the intact galls with a paint brush that would kill crown galls and olive knots without any surgical pre-treatment.

MATERIAL AND METHODS

Experiments on the degree of penetration of a number of organic solvents showed that butyl (normal) and iso-amyl alcohols and kerosene possess a strong affinity for crown gall tissues and penetrate as soon as they are placed on the surface of the gall. These materials were used to move the antibiotics into the galls. In order to render the antibiotics less mobile after they are taken into the tissues, and thus provide more lasting action, small quantities (1/2 percent) of both lanolin and vaseline were added to the mixture.

Preparation of the antibiotic drenches was carried out in the following order: to a saturated solution of a weighed amount of an antibiotic in water, add iso-amyl gradually, followed by kerosene, melted lanolin, and vaseline mixture. If the mixture is properly prepared, it will be

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clear or it will be slightly opalescent, due to water present in either iso-amyl alcohol or kerosene, or extra water in the solvents. This opalescence does not impair the quality of the drench. Before the mixture is used for painting the galls it should be shaken. No turbidity occurred with drenches containing 1000 ppm streptomycin, but occasional turbidity was encountered with drenches containing 1500 or 2000 ppm streptomycin. Terramycin used at 200 ppm gave a slightly opalescent solution. In all experiments crystalline forms of streptomycin and terramycin were used.

Soft galls (on tomatoes) and woody galls on young apricot and peach trees produced by the file method of inoculation described in an earlier paper were used in the experiments. Hundreds of such galls were painted with the experimental drenches. Care was taken to paint the healthy tissue not more than about 1/2 inch beyond the galls. Proper checks painted with iso-amyl alcohol, butyl (normal) alcohol, kerosene, lanolin, and vaseline without the antibiotics were included, as were non-treated galls.

RESULTS

Formulations III, IV, and V gave uniformly good results (Table 1). They all contained iso-amyl alcohol, kerosene, vaseline and lanolin, and streptomycin or terramycin, or both. Of the solvents, kerosene is the most potent against the galls when used without antibiotics. It moves very rapidly in the host and is capable of causing serious injury. Both iso-amyl alcohol and butyl alcohol where used alone in a concentration used in the experiments gave variable results, and in most cases the galls were not destroyed completely. Addition of either lanolin or vaseline appears to reduce the rate of mobility of the solvent, thus confining its activity to the gall.

Table 1. Composition and effect of antibiotic drenches on woody types of crown galls (Agrobacterium tumefaciens) induced on young greenhouse-grown apricot and peach trees, and against olive knot, artificially produced on small greenhouse-grown olive trees.

Formulation	Composition	Parts	Control obtained for[a]: Crown gall on: Apricot	Peach	Olive knot	Remarks
I	Streptomycin Iso-amyl alcohol Kerosene	2000 ppm 20 percent 80 percent	Fair to good	Fair to good	Fair	Occasional injury to tissues beyond treated areas
II	Streptomycin Butyl (normal) alcohol Kerosene	2000 ppm 20 percent 80 percent	Fair	Fair	Poor	Occasional injury to areas beyond treatment
III	Streptomycin Iso-amyl alcohol Kerosene Vaseline Lanolin	2000 ppm 20 percent 80 percent 1/2 percent 1/2 percent	Excellent	Excellent	Excellent	No injury to healthy tissues beyond treated area
IV	Streptomycin Terramycin Iso-amyl alcohol Kerosene Vaseline Lanolin	2000 ppm 200 ppm 20 percent 80 percent 1/2 percent 1/2 percent	Excellent	Excellent	Excellent	Same as in III
V	Terramycin Iso-amyl alcohol Kerosene Vaseline Lanolin	200 ppm 20 percent 80 percent 1/2 percent 1/2 percent	Excellent	Excellent	Excellent	Same as in III

[a]Designations on control: Excellent = 100 percent; fair to good = 30 percent and better; poor = 1 percent and better.

Only a small quantity of the antibiotic drench is required to destroy small to medium size crown galls in only 4 to 5 days.

Olive knots painted with formulations III, IV, and V were completely inactivated within 1 week and no recurrence of the disease was observed after 6 months from the time of treatment when the experimental plants were discarded. The same is true for the crown galls.

DISCUSSION

Attempts to control crown gall by antibiotic sprays possessing some systemic action have not been successful. Brown (5) and Brown and Boyle (6) reported that crude penicillin solutions applied as compresses to crown galls on Bryophyllum and other plants resulted in eventual destruction of the galls. This operation appears impractical. The difficulty in successful control of crown gall lies in the high degree of impermeability of crown gall tissues to aqueous preparations. The elgetol-methanol eradicant drench developed for crown gall (3, 4) gives good results when properly applied. The bad features of this formulation are its strong affinity for cotton and wooly fabrics worn by operators and its lasting staining of the skin. The fact that the ingredients of the antibiotic formulations presented in this paper are not injurious to the skin or the clothing of the operators, and that they penetrate the galls with minimum destruction of healthy plant tissues, seems to make these streptomycin and terramycin drenches preferable to the elgetol-methanol preparation.

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SODIUM USNATE AS AN ANTIBIOTIC FOR PLANT DISEASES

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Abstract

Sodium usnate, an antibiotic from lichens, is effective against the tomato canker organism, Corynebacterium michiganense, but not against gram negative phytopathogenic bacteria such as Agrobacterium tumefaciens, Erwinia amylovora, E. carotovora, Pseudomonas syringae, P. phaseolicola, P. lachrymans, Xanthomonas juglandis, X. vesicatoria, and X. malvacearum. It is toxic to the spores of cucumber and lima bean downy mildews, bean rust, and brown rot of stone fruit. Both spray and dust formulations of the antibiotic gave effective experimental control of downy mildew of cucumber, downy mildew of lima bean, bean rust, bean anthracnose, powdery mildew of bean, and brown rot of stone fruits. Excessive humidity in the presence of the antibiotic causes injury which can be prevented by incorporation of 1 percent chlorophyllin into the spray.

Usnic acid is derived from various lichens. Its isolation dates back to the end of the 19th century. Since that time the substance has been studied chemically and biologically in different centers of the scientific research (3, 4, 5, 7, 9, 12, 13, 17). A detailed account of the subject of usnic acid in its botanical, chemical, and medical aspects is found in a recent book by Lasarev and Savich (8).

Usnic acid is insoluble in water, only slightly soluble in alcohol, petroleum and ethyl ether, and readily soluble in benzene, chloroform, amyl alcohol, and glacial acetic acid. As an antibiotic usnic acid has been reported to be active against gram positive bacteria such as Mycobacterium tuberculosis, Staphylococcus aureus, and others (4, 5, 6, 9, 11, 13, 15, 16, 18). Among plant pathogens it is reported that usnic acid is bactericidal in a concentration of 1 to 400,000 for Corynebacterium michiganense (18); but only a bacteriostatic action was noted at dilutions 1:2500 and 1:3500 against Pseudomonas solanacearum, Erwinia carotovora, and Xanthomonas phaseoli, and there was no effect on X. translucens (8). Tests against Aspergillus sp., Mucor sp., and Penicillium sp. appeared to be negative (8).

Although the literature gives a general idea of the preparation of biologically active sodium usnate (8, 10), it is lacking in detail and is too general. Therefore, after some experimentation a method was devised for the preparation of biologically active sodium usnate (Bottini). The method is simple and can be performed with success in any laboratory. This paper reports on the action of sodium usnate prepared from usnic acid[4] on certain plant pathogens.

METHOD

The sodium usnate was prepared using the principle reported by Lasarev and Savich (8). A solution of 20 grams of anhydrous sodium carbonate and 1 liter of distilled water was prepared in a 3-liter flask equipped with a mechanical stirrer. One liter of ethanol and 20 grams of usnic acid were added and the resulting mixture was stirred until it became homogeneous (about 4 hours). The mixture was divided into four 500-ml portions and each portion was rapidly concentrated to 200 ml on a rotary film evaporator at 50° C and aspirator pressure. Crystals were noted in each concentrated solution when the volume was reduced to about 250 ml. The concentrates were combined and set at 0° C overnight. The light yellow crystals were collected by suction filtration and dried in a vacuum desiccator. The yield was 22.7 grams, 98 percent based on formation of sodium usnate dehydrate ($C_{18}H_{15}O_7Na.2H_2O$). This material was used

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for both *in vitro* and *in vivo* studies as aqueous solutions and dust formulations.

The bacterial organisms for *in vitro* tests were grown on potato-dextrose-peptone agar and the fungi on potato-dextrose agar. Spores of the downy mildews and rust were obtained from greenhouse grown plants and germinated in water or in some cases on water agar. Filter paper discs (S and S, 740E) were employed in toxicity tests on bacteria and fungi *in vitro*.

The experiments *in vivo* were done in a warm greenhouse (70° to 80° F) with a relative humidity varying from 70 to 80 percent. As a rule all inoculated plants were held in a moist chamber for 18 hours after inoculation.

Infection on plants inoculated with cucumber downy mildew (*Pseudoperonospora cubensis*) and bean rust (*Uromyces phaseoli*) was evaluated by counting the number of lesions, while for brown rot of stone fruits (*Monilinia fructicola*), bean anthracnose (*Colletotrichum lindemuthianum*), and cucumber scab (*Cladosporium cucumerinum*), the disease was expressed in terms of relative severity. The Bountiful and Pinto varieties of beans were used for bean anthracnose, powdery mildew (*Erysiphe polygoni*) and rust. Lima bean (Fordhook var. #242) was employed for downy mildew of lima bean (*Phytophthora phaseoli*), and apricot was used as the brown rot host.

The stability of sodium usnate was tested by (a) exposing the liquid preparation to light at room temperature for an extended period of time, and (b) by storing dust formulations at room temperature and at 65° C for 3 weeks; and afterwards bioassaying with *Corynebacterium michiganense* by a filter paper disc method (1).

RESULTS

Tests against the phytopathogenic bacteria showed that sodium usnate was active only against gram positive bacteria as represented by *Corynebacterium michiganense*. The tests were negative for *Erwinia amylovora*, *E. carotovora*, *E. aroideae*, *Xanthomonas juglandis*, *X. malvacearum*, *Pseudomonas phaseolicola*, *P. syringae*, *P. lachrymans*, and *Agrobacterium tumefaciens*.

The conidia of downy mildew of cucumber appeared to be more sensitive to sodium usnate than did those of lima bean mildew as determined by a slide method. Thus, while the conidia of both types of mildew perished in a concentration of sodium usnate containing 25 ppm, the difference was apparent at lower concentrations. At 10 ppm of sodium usnate the conidia of downy mildew of cucumber did not germinate, and at 2 ppm only 2 percent of the conidia germinated, with 96 percent germination in the check. The conidia of downy mildew of lima bean gave 29 percent germination in the 2 ppm solution, 0.33 percent in 10 ppm, and 27 percent in water (check). The germination of spores of bean rust and brown rot in the presence of sodium usnate was as follows: Bean rust: check--76 percent, sodium usnate 1 ppm--64 percent, 2 ppm--37 percent, 5 ppm-- 8 percent, 10 ppm-- 1 percent, 25 ppm-- 0 percent; Brown rot: check--99 percent, sodium usnate 1 ppm-- 43 percent, 2 ppm-- 32 percent, 5 ppm-- 39 percent, 10 ppm-- 33 percent, 25 ppm-- 7 percent, and 50 ppm-- 0 percent.

The experiments on controlling various plant pathogens under the greenhouse conditions showed that downy mildew of cucumber was not controlled by the antibiotic spray at concentrations of sodium usnate below 100 ppm. Downy mildew of lima bean was more resistant to the action of the antibiotic and showed good results only when the spray contained 500 ppm sodium usnate. Bean rust responded to the antibiotic fairly well. A water-sprayed check showed 109 pustules per leaf, while for leaves sprayed with 100, 200, and 500 ppm sodium usnate there were only 3.7, 1.5, and 0.3 pustules per leaf, respectively.

For bean anthracnose the effective spray contained 300 ppm sodium usnate and above, although at times fairly good control was obtainable with 200 ppm. Control of brown rot of stone fruits with the antibiotic sprays of sodium usnate was not uniformly satisfactory, although in some cases a complete protection was obtainable with 500 ppm. No control of cucumber scab was observable with concentrations of 300, 500, and 800 ppm of sodium usnate used in the experiments. The powdery mildews of cucumber and bean were controlled by sodium usnate, bean powdery mildew being the more sensitive to the antibiotic, which was effective at 200 ppm. No control of cucumber powdery mildew was obtained with 500 ppm sodium usnate.

While spray formulations of sodium usnate yielded variable results for different pathogens and different hosts, dust formulations consisting of sodium usnate in a nuclay pyrophyllite carrier gave remarkable control in some experimental plant diseases. Complete prevention of downy mildew of cucumber and bean rust was accomplished by dust formulations containing 1000 and 2000 ppm of sodium usnate antibiotic. Fairly good control was observed at 500 ppm. Bean anthracnose was controlled completely with 2000 ppm dust formulation and fairly well with

1000 ppm. Brown rot of apricot responded well to the dust containing 2000 ppm but not to 1000 ppm.

The sodium usnate antibiotic is capable of causing injury on plant surfaces of beans and cucumbers when treated plants are subject to prolonged action of high humidity (80 percent relative humidity or higher). The injury expressed itself in dead spots and can be severe. It is of interest to note that this injury was prevented when 1 percent chlorophyllin was added to the mixture, behaving in this respect as it does with other antibiotics such as streptomycin, Actidione, and so forth (2). The injury is not associated with the pH of the antibiotic since it was 7.4 for both 300- and 500 ppm.

Sodium usnate preparations are stable in sprays and dusts. The dust formulations held at 65° C for 3 weeks did not lose their activity when tested on plants against Corynebacterium michiganense, a standard test organism for this antibiotic.

No systemic action in the plant could be demonstrated when the antibiotic was introduced into the cucumber and bean plants through the roots or leaf surfaces.

DISCUSSION AND CONCLUSION

Sodium usnate prepared from usnic acid, an antibiotic contained in many lichens, may be useful in preventing certain diseases of plants. Although its action against phytopathogenic bacteria is mainly confined to a small group of gram positive organisms such as tomato canker, its marked activity against downy mildews, rusts, some powdery mildews and other fungi should make it a desirable new addition to the remedies for diseases of plants. That sodium usnate does not seem to become systemic in treated plants should be a desirable feature from the standpoint of residue tolerances set by the Pure Food and Drug Administration for many agricultural poisons.

The pharmacology of sodium usnate is known from the work of Snegirev (14), who showed that the LN50 for white mice in intraperitoneal injections equals 49 mg/klg, and 600 mg/klg when administered through the stomach of experimental rabbits. These data indicate the considerable toxicity of sodium usnate when introduced by the methods indicated. However, there appears to be no appreciable absorption of the antibiotic through the skin or through wounds. The antibiotic is used in the U.S.S.R. under the name Binan, and is recommended for treatment of fresh wounds, varicose and trophic ulcers, plastic surgery, second and third degree burns, and many other conditions (8). Thus, as an agricultural antibiotic, it should not meet with serious objections.

Since lichens are abundant and since the production of usnic acid and sodium usnate is a relatively simple and inexpensive procedure, it might be profitable to investigate the therapeutic possibilities of lichens further, not only from the standpoint of usnic acid but also from that of many other antibiotics contained in them (5, 6, 13, 15, 16).

On the basis of the tests presented in this paper one may conclude that sodium usnate is effective against Corynebacterium michiganense (in vitro) and downy mildews of cucumber and lima beans, bean rust and brown rot of stone fruits (both in vitro and in vivo). The effective concentrations may vary with the pathogen and the host. The antibiotic sprays were especially effective against downy mildew of cucumbers, bean rust, and powdery mildew of bean. Dust formulations containing 1000 and 2000 ppm sodium usnate gave excellent control of downy mildew of cucumber, bean rust, and bean anthracnose. In general, dust formulations performed better than the sprays. Plant injury from sodium usnate develops on bean and cucumber plants under excessive humidity but can be prevented by the addition of 1 percent chlorophyllin.

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HYDRATED LIME IMPROVES NUCLAY-STREPTOMYCIN DUST FOR PEAR BLIGHT CONTROL

Peter A. Ark and James P. Thompson[1]

Abstract

The addition of hydrated lime to the Nuclay-streptomycin dust formulation greatly improves physical and microcidal properties and its usefulness in control of fire blight of pear.

It has been shown that streptomycin dust, when formulated in Nuclay pyrophyllite and properly applied to the trees, gives satisfactory control of fire blight (Erwinia amylovora) in pear (1), and the downy mildews of lima bean (5) as well as of cucumber, radish, and spinach (authors' unpublished data).

Sometimes the users of either a Nuclay or Pyrax ABB streptomycin dust formulation state that the dust does not seem to adhere well to dusted leaf surfaces and thus some activity of the antibiotic may be lost. Therefore, the addition to such dusts of some suitable carrier that would be compatible with the antibiotic and provide fluffiness and better adherence to the leaf surface would be a welcome improvement. Many commercially used carriers, such as frinite, bentonite, attaclay, and talc cannot be recommended since they absorb streptomycin irreversibly (2). Calcium carbonate, magnesium carbonate, and calcium phosphate are excellent carriers for streptomycin and can lighten both Nuclay and Pyrax ABB dusts, but their cost is high. However, hydrated lime, which is not expensive and possesses many desirable features as a carrier for various plant medications, may be considered as a safe and satisfactory carrier for streptomycin. In spite of its high alkalinity, hydrated lime does not affect the antibiotic adversely. It is fluffy and light and when placed on a moist leaf surface will adhere to it firmly, resisting both strong wind and heavy rain. The rate of release of streptomycin from hydrated lime is about the same as that from Nuclay particles. The pH value of a hydrated lime-streptomycin formulation is higher than that of a Nuclay-streptomycin formulation, and this appears to improve the solubility and activity of streptomycin against susceptible microorganisms.

The tests (Table 1) show that adding 50 percent hydrated lime to the Nuclay dust enhances its microcidal potential by about 11 percent, as indicated by the width of the inhibition zones around filter paper discs placed on agar plates seeded with Erwinia amylovora.

Since hydrated lime lightens the dust, it is possible to use smaller quantities of the antibiotic per application and thus reduce the cost of each application. Reduced cost of dust per-

Table 1. Activity of Nuclay, hydrated lime, and Nuclay-hydrated lime streptomycin (1000 ppm) dust formulations against Erwinia amylovora and the pH values of the formulations.

Carrier plus streptomycin (1000 ppm)	Inhibition zone (mm)[a]	pH[b] Paste	pH[b] Streptomycin solution
Nuclay	27	8.2	8.7
Hydrated Lime (H.L.)	30.8	10.2	10.2
Nuclay + 2 percent H.L.	27	8.0	8.4
Nuclay + 50 percent H.L.	30	10.0	10.1

[a]Activity against the bacteria is expressed as inhibition zones (mm) produced by discs (S & S-740E) dipped in streptomycin solution (100 ppm) derived from the dust formulations and placed on nutrient agar plates with subsequent incubation at 28° C.
[b] The pH values measured from paste, and streptomycin solution (100 ppm) prepared from dusts.

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mits the more frequent applications which may be necessary for proper protection from fire blight in pear or apple. Fire blight epidemics in the so-called dry spring and summer years are due to light or heavy dews prevalent in orchards at night or in the early morning hours. During such seasons there is an urgent need for a dependable bactericide to prevent the infection from which an epidemic can result. Streptomycin dust made by the addition of hydrated-lime may answer this need since it adheres well on surfaces wet with dew. The importance of dew has been emphasized for many bacterial and fungal diseases by numerous investigators (3, 4, 6).

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DOWNY MILDEW OF SMALL GRAINS AND GRASSES IN ARKANSAS[1]

J. L. Dale and G. E. Templeton[2]

During the spring and summer of 1959 downy mildew, incited by Sclerophthora macrospora (Sacc.) Thirum., Shaw, & Naras., was observed in Arkansas on barley, oats, rye, wheat, fescue, and goosegrass.

The disease was found in severe proportions on the small grains at the Rice Branch Experiment Station at Stuttgart. Major damage occurred on oats, with local restriction of the disease to low, poorly drained areas which had been flooded at some time during the growing season. Diseased plants were found, however, scattered at random throughout fields. Both fall-and spring-planted oats were affected, and oats grown from seed that had been treated with a mercurial seed treatment also suffered from the disease. All plantings were on ground that had not been planted to oats for three preceding years.

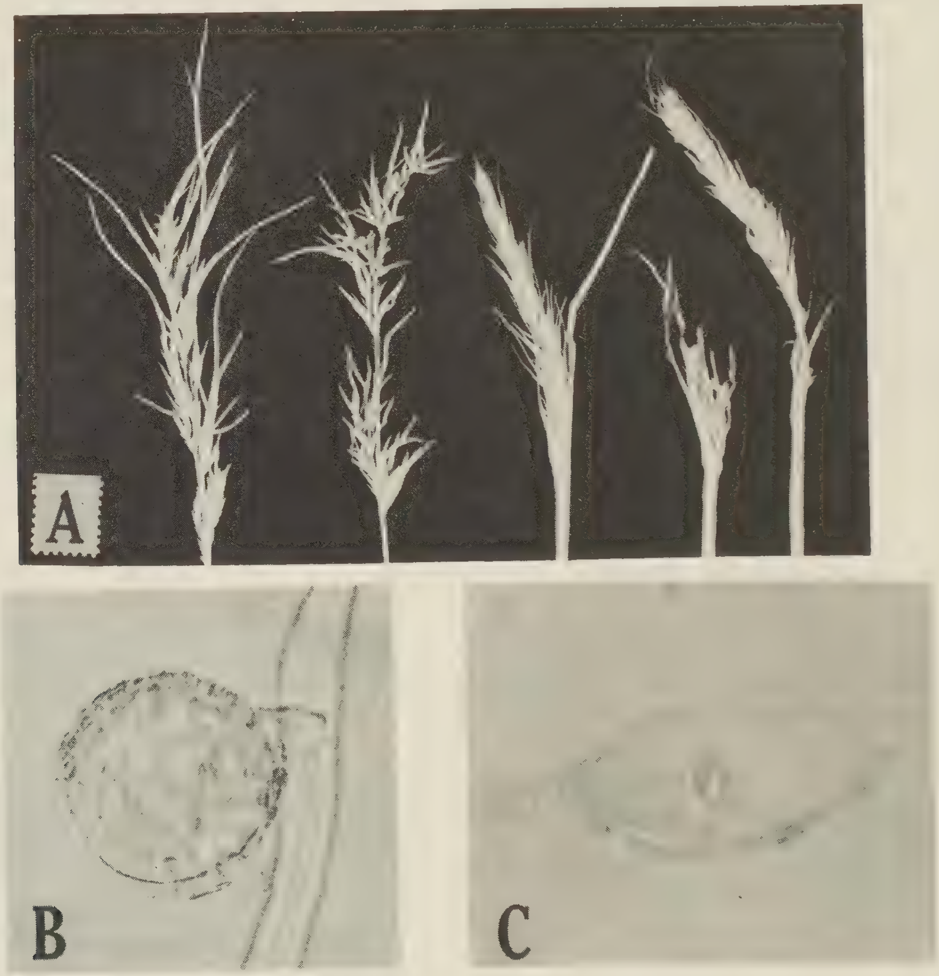

FIGURE 1. Symptoms of downy mildew on fescue and rye inflorescences, and oospores from rye tissue. A -- Mildew symptoms on fescue (left) and rye (right) inflorescences. B -- Typical oospore from rye tissue. C -- Oval-shaped oospore from rye tissue.

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One affected area extended across a purity-trial planting made by the Arkansas State Plant Board and provided an opportunity to observe the effects of the disease on barley, rye, and wheat. This was the first time the disease had been observed in the State on these small grains and, from an investigation of the literature, it appears to be the first time S. macrospora has been reported on rye (Secale cereale) in the United States. It was reported previously on oats in the State by Summers, Adair and Stanton (5).

The symptoms on rye most closely resembled those observed on barley. Distortion and proliferation were less striking than that reported on corn, wheat, oats, and many grasses (1, 3, 4, 6). Rye plants were stunted, and distortion of the awns was more frequently observed than gross distortion of the other parts (Fig. 1).

Downy mildew occurred on tall fescue, Festuca arundinacea, to some extent in most areas of Arkansas where fescue is used as a pasture grass. The disease was very prevalent in one large pasture in the White River bottoms in northwest Arkansas, and occurred to a lesser extent in pastures in the Red River bottoms and Coastal Plains in the southwest part of the State, in the east-central section in the Mississippi Delta area, and in the north-central section in the Ozark Highlands. The disease was found on goosegrass, Eleusine indica, in only one locality in southwest Arkansas.

The disease on fescue in Arkansas was more generally distributed than noted in the only previous report of it on fescue in the United States (2). The pastures containing the diseased fescue generally were in locations subject to prolonged wet periods, except in the Ozark Highlands where diseased plants were found in a hillside pasture in rocky, apparently well-drained soil.

Oospores in tissue of rye cleared in NaOH were spherical to oblong, smooth, and a measurement of 200 spores yielded a range in spore size of 41 to 113μ, with a mean size of 56 x 69μ. Measurement of 200 oospores in cleared stem tissue of the fescue revealed a mean spore diameter, or length, of 57μ, the range being 31-98μ. There were some distinctly oval-shaped oospores in the fescue, and in the rye tissue such spores were very numerous. No sporangia were observed. Both typical and oval-shaped oospores from rye tissue, and symptoms of the disease on rye and fescue inflorescences are shown in Figure 1.

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CONTROL OF LEAF AND STEM RUST OF WHEAT BY ZINEB AND INORGANIC NICKEL SALTS[1]

F. R. Forsyth and B. Peturson[2]

Summary

Nickel salts, with and without zineb, were applied to control leaf and stem rust of wheat on small field plots of Thatcher, Marquis and Red Bobs wheat at Winnipeg in 1959. Economic control was obtained with all combinations of nickel and zineb used, the greatest net gain resulting from the use of zineb as a protectant at the rate of 2 1/2 pounds per acre with six applications. Benefits resulting from the use of zineb plus nickel salts on an eradicative schedule were not in direct proportion to the increased yields, due to the fact that the zineb component of the mixture is considerably more expensive than the nickel. Nickel sulfate at rates of 1 to 1 1/2 pounds per acre, with 7-day intervals between applications seems to be an efficient eradicative fungicide of leaf and stem rust on Thatcher, Marquis and Red Bobs.

Experimental results at Winnipeg in 1957 and 1958 (3) indicated that the inorganic salts of nickel, particularly the chloride and nitrate, could be used to control leaf rust, Puccinia recondita Rob. ex. Desm. f. sp. tritici (=Puccinia rubigo-vera (DC.) Wint. f. sp. tritici (Eriks.) Carleton) on Thatcher wheat in the field. The increased yields resulting from the application of inorganic nickel were not as great as those from the use of the protective fungicide zineb. Consequently small field plot tests were conducted in 1959 to attempt to confirm the results of 1957 and 1958, to extend them to other wheat varieties, and to ascertain whether or not this effect can be expected if both leaf and stem rust (Puccinia graminis f. sp. tritici Eriks. and Henn.) are present. Various combinations of nickel salts and zineb were used in a search for the most efficient and economical method of controlling the rusts.

METHODS

The varieties of wheat Thatcher, Marquis and Red Bobs were used in the 1959 tests. It was expected that leaf rust of wheat would be present on the three varieties to about the same degree. However, the reactions of the hosts to stem rust might be quite different since Red Bobs has little or no resistance to stem rust, Marquis has some tolerance to certain races, and Thatcher is resistant to most races of stem rust except 15B.

The wheat was grown in rows 1 foot apart in plots 4 feet by 18 feet with four buffer rows between test plots. There were four replicated plots of each check and fungicidal treatment. The locations of the plots within a block were chosen at random, each block containing one plot of each treatment and one check plot.

Infection by stem and leaf rust was due entirely to naturally occurring inoculum. The results of this year's tests are the most interesting because Thatcher did not have any stem rust, no doubt because of the very low level of 15B inoculum in the air at Winnipeg in 1959. Stem rust infection developed slowly on Marquis and very rapidly on Red Bobs. Leaf rust developed on all three varieties of wheat.

The fungicides were applied with knapsack sprayers of 1-gallon capacity and with a pressure of 40 p.s.i. A weighed amount of fungicide was applied to each plot in 500 ml of water containing four drops Triton X-114 as a spreader-sticker. This is equivalent to 66 gallons of water per acre.

The criteria used to evaluate the effectiveness of the chemicals under test were: the percentage of rust at the final reading by use of the modified Cobb's scale (4); the weight of 1000 kernels; the yield in bushels per acre; the weight per bushel in pounds; and the increased net gain in dollars per acre attributable to the use of the fungicide.

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Nickel sulfate hexahydrate was tested in addition to nickel chloride and nickel nitrate this year because the sulfate is less expensive than the other two nickel salts. It is produced in Canada as a by-product of Canadian mining operations. The fungicides were applied on an eradicative schedule, at approximately 7-day intervals, commencing on July 10th when the plants were at the flag leaf stage and 80 percent of the leaves immediately below the flag leaves had from 0 to 10 percent leaf rust. A treated check was included to give a measure of the yield of each variety under test in the virtual absence of the rust. Zineb was used for this purpose on a protective schedule at the rate of 2 1/2 pounds per acre compared with the lower rates used for the treatments with combined zineb and nickel salts.

RESULTS

The results of the applications of nickel salts, zineb, and nickel plus zineb to the three varieties of wheat are presented in Table 1. Plots of all three varieties, treated with six applications of zineb (65 percent W.P.) at 2 1/2 pounds per acre as a protective fungicide gave higher yields per acre and greater 1000 kernel weights than did any of the other treatments. In some instances treatment with nickel chloride plus zineb resulted in greater weights per bushel than did zineb alone.

The amounts of nickel used in these tests did not cause serious plant injury. This lack of phytotoxicity is in part attributable to the fact that no solutions of nickel were applied before the flag leaf stage, by which time there is considerable resistance to nickel.

The percentage of leaf rust and of stem rust on the plants after the various treatments should be a good indication of the effectiveness of the fungicide. However, there was not a direct relationship between rust percentage and yield, especially in the plots treated with nickel or with nickel plus zineb. The percentages of rust recorded for all of the nickel treated plots were low. A possible reason for this is that each application of nickel kills and blackens the rust pustules present. These blackened areas vary in size and are difficult to take into consideration when estimating the percentage of the leaf surface covered by rust. The rust readings thus may not be a true indication of the total number of infections present on the leaf.

The partial or complete control of the rust must be accompanied by a sufficient increase in yield and quality to justify the expense involved. Table 2 lists the net gain per acre resulting from the use of nickel salts and zineb to control leaf and stem rust of wheat. The cost of application was not taken into consideration owing to lack of precise information on spraying costs. However it is estimated that the cost of spraying with ground equipment would be about 30 cents per acre per application. The increased returns per acre from the crop after deducting the cost of the fungicide are appreciable in most instances. The use of zineb (65 percent W.P.) at a rate of 2 pounds per acre on a 7-day schedule and with only four applications gave the poorest return per acre. On the other hand, when the zineb was used in combination with nickel salts the effectiveness of both zineb and nickel was enhanced.

DISCUSSION AND CONCLUSIONS

Forsyth and Peturson (1) stated that economical control of stem and leaf rust of wheat could be achieved by the use of zineb as a protective fungicide provided that the amount and timing of rainfall was not adverse during the spray program. It was extremely important to begin application of the chemical early, when only a trace of rust was present. The climatic conditions in 1959 at Winnipeg were such that economic control was achieved in small plots by the use of six applications of zineb at the rate of 2 1/2 pounds per acre. There was rain within 24 hours of application of zineb on only one occasion, July 9. The fungicide was applied again within 3 days. This again demonstrates how effective zineb can be in controlling rust when the rainfall is not excessive.

The increase in yield obtained by spraying plots six times, with 2 1/2 pounds of zineb per acre, was very much greater than when zineb was applied four times at 2 pounds per acre. The difference in yield, resulting from much better control of rust, could not be attributed to the extra 1/2 pound of zineb applied on each date. No significant increase in either rust control or yield was obtained from plots treated with nickel chloride plus 1 1/2 pounds of zineb compared with those given nickel chloride plus 1 pound zineb. The difference is almost certainly attributable to the use of two additional applications made and to their timing. The two extra applications were made 10 and 2 days, respectively, before the other spray treatments were started. By the time that the four application program was started, appreciable amounts of leaf rust had developed on the lower leaves of the wheat plants. This very great importance

Table 1. Result of applications of nickel salts, zineb and nickel plus zineb for rust control on field plots of wheat at Winnipeg, 1959.

Treatment	Dates of application	Pounds per acre	Number of applications	Percentage leaf rust	Percentage stem rust	Yield of check in bu./acre and diff. from check	Average 1000 kernel weight of check and diff. from check	Weight of check per bushel (pounds) and diff. from check
Thatcher wheat								
				July 27				
Check			0	95.0	zero	13.2	19.09	56.75
Zineb 65% W.P. (Treated check)	June 30, July 8, 11, 17, 24, 30	2 1/2	6	3.0	do	+30.2	+10.42	+4.30
Nickel chloride pluz zineb	July 10, 17, 24, 31	1 1/2 plus 1 1/2	4	6.3	do	+21.6	+7.63	+3.14
Nickel chloride plus zineb	do	1 plus 1 1/2	4	6.3	do	+19.6	+8.25	+4.44
Nickel chloride plus zineb	do	1 plus 1	4	8.0	do	+18.7	+7.78	+4.12
Nickel sulfate plus zineb	do	1 plus 1 1/2	4	7.5	do	+18.6	+7.62	+4.65
Nickel nitrate	do	1	4	9.3	do	+15.6	+5.47	+3.50
Nickel chloride	do	1	4	13.7	do	+14.4	+5.85	+1.39
Nickel sulfate	do	1	4	15.0	do	+14.2	+5.15	+2.69
Nickel chloride	do	2	4	8.5	do	+11.5	+5.40	+2.81
Zineb 65%	do	2	4	42.5	do	+11.5	+5.65	+3.25
					L.S.D. 5%	5.3	1.08	1.45
					L.S.D. 1%	7.2	1.85	2.49
Marquis wheat								
				July 27	Aug. 11			
Check			0	75.0	52.5	12.1	19.15	54.50
Zineb 65% W.P. (Treated check)	June 30, July 8, 11, 17, 24, 30	2 1/2	6	2.0	2.8	+25.1	+14.81	+6.31
Nickel sulfate plus zineb	July 10, 17, 24, 31	1 plus 1 1/2	4	4.5	20.0	+14.9	+8.74	+5.00
Nickel chloride plus zineb	do	1 plus 1	4	4.8	17.5	+14.5	+8.52	+4.81
Nickel chloride plus zineb	do	1 plus 1 1/2	4	5.0	12.5	+13.2	+11.60	+5.75
Nickel chloride plus zineb	do	1 1/2 plus 1 1/2	4	3.5	12.0	+12.0	+8.42	+5.44
Nickel chloride	do	1	4	5.5	22.5	+11.2	+7.13	+3.81
Nickel chloride	do	1 1/2	4	4.5	18.8	+9.7	+6.70	+3.76
Nickel sulfate	do	1	4	7.3	25.0	+8.8	+6.08	+3.44
Zineb 65%	do	2	4	26.3	22.5	+7.4	+6.48	+4.20
Nickel nitrate	do	1	4	8.8	20.5	+7.0	+6.98	+3.81
					L.S.D. 5%	3.6	2.22	1.02
					L.S.D. 1%	4.9	2.99	1.37
Red Bobs wheat								
				July 28	Aug. 11			
Check			0	87.5	72.5	2.7	11.13	39.0
Zineb 65% W.P. (Treated check)	June 30, July 8, 11, 17, 24, 30	2 1/2	6	7.8	18.8	+16.6	+12.22	+13.8
Nickel chloride plus zineb	July 11, 18, 25, 31	1 plus 1 1/2	4	11.3	36.2	+13.3	+10.23	+13.5
Nickel chloride plus zineb	do	1 1/2 plus 1 1/2	4	10.0	36.2	+12.4	+11.33	+14.5
Nickel chloride plus zineb	do	1 plus 1	4	18.8	38.8	+9.5	+7.73	+10.5
Nickel sulfate plus zineb	do	1 plus 1 1/2	4	12.5	43.8	+9.3	+9.62	+11.5
Nickel chloride	do	1 1/2	4	12.5	47.5	+9.1	+8.07	+10.3
Nickel chloride	do	1	4	22.5	48.8	+7.8	+6.33	+8.7
Nickel sulfate	do	1	4	18.8	50.0	+7.3	+6.20	+9.7
Nickel nitrate	do	1	4	20.0	43.8	+5.4	+6.53	+10.3
Zineb 65%	do	2	4	55.0	48.8	+5.1	+3.79	+6.5
					L.S.D. 5%	3.6	1.98	2.29
					L.S.D. 1%	4.8	2.69	3.09

Table 2. Net returns[a] per acre resulting from use of nickel salts and zineb to control leaf and stem rust of wheat.

Fungicide treatment	Rate (pounds per acre)	Number of applications	Cost of fungicide	Increased value of the crop per acre after deducting cost of fungicide		
				Thatcher	Marquis	Red Bobs
Zineb 65% W.P. (Treated check)	2 1/2	6	$11.82	$38.01	$29.60	$15.57
Nickel sulfate plus zineb	1 and 1 1/2	4	6.32	24.37	18.27	9.03
Nickel chloride plus zineb	1 and 1	4	5.08	25.78	18.85	10.60
Nickel chloride plus zineb	1 and 1 1/2	4	6.64	25.70	15.14	15.31
Nickel chloride plus zineb	1 1/2 and 1 1/2	4	7.60	28.04	12.20	12.86
Nickel chloride	1	4	1.92	21.84	16.56	10.95
Nickel chloride	1 1/2	4	2.88		13.13	12.13
Nickel chloride	2	4	3.84	15.14		
Nickel sulfate	1	4	1.60	21.83	12.92	10.45
Zineb 65% W.P.	2	4	6.32	12.66	5.89	2.10
Nickel nitrate	1	4	1.92	23.82	9.63	6.99

[a] Based on a value of $1.65 per bushel of wheat, 79¢ per pound of zineb 65 percent (in this case Dithane Z-78), 48¢ per pound of nickel chloride hexahydrate, 40¢ per pound of nickel sulfate hexahydrate and 48¢ per pound of nickel nitrate. The cost of application was not taken into consideration.

of timing when using a protectant fungicide such as zineb illustrates its greatest weakness (1). The effectiveness of the spray program becomes almost entirely dependent on success in applying the chemical at the critical time in relation to rust infection and rainfall.

Eradicant chemicals such as the nickel salts (2, 3) are not influenced to the same extent by rainfall. As they act on the rust infections that are present at the time of application, rainfall a day or so after treatment does not diminish their effect. The experimental results obtained in any one year are therefore likely to be valid in most seasons when rust infection is heavy enough to be injurious.

The three nickel salts tested all appeared to be effective in controlling leaf rust, and to be fairly effective against stem rust, when applied on an eradicative schedule at 7-day intervals. The yields per acre, weight per 1000 kernels, and weight per bushel of wheat from treated plots were greater than those from the untreated check plots. Much more important to wheat growers, however, is the fact that net returns per acre, after deducting the cost of the chemicals used, were much greater from plots given effective treatments than from the rusted check plots.

Nickel sulfate appeared to be less phytotoxic than nickel chloride to wheat. As the sulfate is equally effective in controlling rust, it would be preferred over both the chloride and the somewhat less effective nitrate because of its lower price and availability in Canada.

The addition of zineb to the nickel salts on an eradicative schedule increases their effectiveness to a certain extent. The higher cost of zineb than of nickel salts, however, reduces the net gain obtained by its use to a level which is not significantly greater than that obtained with nickel salts alone.

The differences in susceptibility to stem rust of the three wheat varieties were reflected in the net gains obtained by spraying them. Thatcher, which was free of stem rust in these tests, gave the highest net gain as the result of chemical treatment. Marquis, which is somewhat tolerant to the races of stem rust present in 1959, gave much better economic returns from spraying than did Red Bobs, which is completely susceptible to stem rust.

It is much more difficult to control stem rust than leaf rust with zineb or with nickel salts. The control obtainable is economically important, however, and if our resistant varieties of wheat should be attacked by new races of rust, the use of nickel salts may avert disastrous losses.

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GLOEOSPORIUM ROT OF STRAWBERRY FRUIT

W. R. Wright, M. A. Smith, G. B. Ramsey, and L. Beraha[1]

Abstract

The occurrence of Gloeosporium sp. as a strawberry fruit rot is reported. This is believed by the authors to be the first such record in the United States.

In May 1958 a truck shipment of strawberries originating from Amite, Louisiana showed approximately 2 percent of the berries affected with brown to dark brown, slightly sunken, circular areas ranging up to 1/2 inch in diameter upon arrival in Chicago (Fig. 1). A fungus characteristic of Gloeosporium was isolated from the rather firm, brown tissue under the lesions. Few references to Gloeosporium as a strawberry pathogen were found. Gloeosporium fragariae (Lib.) Mont. is recorded as a cause of leaf spotting (3). Bennett (1) noted a Gloeosporium sp. associated with the black root-rot of strawberry. The only references to Gloeosporium as a rot of strawberry fruit are those of Sturgess (4, 5), who found it on late-season preserve strawberries in Queensland. Of the published descriptions only that of Sturgess indicates a possible similarity to the fungus described here.

Mycelium of the fungus on potato-dextrose agar is white to light grey darkening to dusky neutral grey and with an early appearance of salmon-colored spore masses, which influence the color of the substrate of the media (2). The septate hyphae range from 2-10 μ in diameter, with heavy, short-coupled, reddish-brown to brown elements mingled with more slender hyphae. Spores are borne either terminally on conidiophores, or in acervuli. Conidiophores range from short to flexuous elements approaching 100 μ in length.

Acervuli range from 40-300 μ in diameter on the host. Conidiophores are seldom over 20 μ long, simple, tending to flare at the base and constricting sharply at the point of attachment to the spores. The spores are continuous, oblong, and rounded at both ends or slightly tapered at one end. They are heavily granular and usually exhibit a central nucleus (Fig. 2).

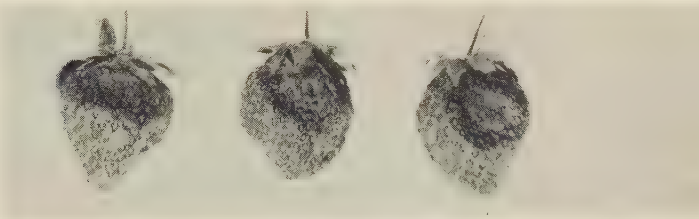

FIGURE 1. Natural infection of strawberries by Gloeosporium sp.

FIGURE 2. Spores of the strawberry Gloeosporium.

Spores measure 3-5μ x 9-19μ (av. 4. 8μ x 14μ). Spores germinate readily in tap water after 5 hours at room temperature by unipolar germ tubes. The fungus grows well on potato-dextrose, corn meal, asparagus, oatmeal, bean and lima bean agars.

Wound inoculations on apple, orange, watermelon, and blueberry fruits were positive. Infection was slight on garlic cloves. Non-wound inoculations of the above hosts, with the exception of blueberries, were negative. Blueberries were readily infected at the stem end, suggesting a tissue weakness, probably due to harvest injury. Inoculations with spore suspensions on ripe strawberry fruits were positive on wounded or sound fruit. Neither setae nor a perfect stage were observed on any media or hosts.

Studies on potato-dextrose agar indicate a minimum growth at 45°, opimum growth at 80°, and a maximum growth at about 97° F.

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Unusually wet weather prior to and during the 1958 picking season apparently provided optimum conditions for this fungus on strawberry fruit. It is not known whether the organism is new to the host in the United States, or whether it may have been present for some time in an unobserved minor role and was able to attack the fruit because of the unusually favorable weather conditions existing that season.

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A SOFT ROOT-ROT OF LINUM USITATISSIMUM CAUSED BY A PSEUDOMONAS SP.

A. Seaman[1]

Abstract

An outbreak of root-rot in flax seedlings (Linum usitatissimum), caused by a Pseudomonas resembling P. fluorescens, was controlled by the use of tetracycline.

INTRODUCTION

During a study of germination extracts of Linum usitatissimum (in collaborative work between the Agricultural Research Council of Great Britain, Unit of Plant Cell Physiology, and the Departments of Chemistry and Horticulture at the University of Nottingham) a root infection of the seedlings became established on such a scale that production of the extract was stopped.

The seed was germinated in aluminum bowls, 2 feet in diameter, across which and one third of the way down expanded aluminum screens were fitted. On these was spread a thin layer of glass wool to support the seeds. The lower space was filled with water; the seed was scattered evenly from a coffee scoop measure, and a plate glass disc covered the bowl. The bowls were then placed in piles (up to 6) in an incubator room fitted with forced draught.

NATURAL INFECTION

In the affected bowls the infection was normally apparent on and after the third day. The radicles which were longer than 0.5 mm turned brown and slimy; in the majority of seedlings this started at a point behind the tip. The germination rate was also poor; many seeds showed a fawn-coloured rot destroying the emergent radicle. By the seventh to ninth day the affected bowls showed many rotted roots and seeds, a few elongated cotyledons, and a heavy overgrowth of a mucor-type fungus.

EXAMINATION OF MATERIAL

Smears from different regions of infected seedlings showed a mixed flora at the root tip and testa, with a small gram-negative rod predominating. The mid-root rot area showed a practically pure culture of small gram-negative rods. A squash preparation showed that no organized cortex was left, the area being occupied by the bacteria. The xylem strands apparently were little affected.

CAUSATIVE AGENT

Pure cultures of the bacterium were readily obtained from roots, radicle and testa by isolation on nutrient agar. On nutrient agar the colonies were small, translucent, entire; growth in broth was flaky, with formation of a pellicle, turbidity and a green pigment. Growth was good at both 22° and 37° C. Glucose was fermented to give acid, but not gas. Lactose was not fermented, starch not hydrolysed, gelatin not liquefied, and H_2S not produced. Nitrate was completely reduced with liberation of ammonia. Zeitnow's stain showed a single polar flagellum. The organism was therefore assigned to the genus Pseudomonas.

Seeds were germinated on filter paper over wet cotton wool in Petri dishes and after 4 days the seedlings were inoculated with 5 ml of a 6-hour (37° C) broth culture. Both inoculated dishes contained badly rotted seedlings after 24 hours, while the control dish showed no rotting after 8 days. The organisms were easily recovered from the experimentally infected seedlings.

SOURCE OF INFECTION

Control measures had already been taken before the bacteriological work was begun. The aluminum bowls were being sterilized in a hot air oven, as were the expanded screens and the

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glass wool. The water had been taken from various sources (tap, demineralizer and distilled) and different supplies of seed had been tried. Seed and equipment from Nottingham did not show any infection when used in a Cambridge Laboratory, but swabs of the walls and floor slats of the incubator room at the University of Nottingham School of Agriculture failed to grow any colonies of similar appearance to the pseudomonad. When the procedure was checked, it was found that the glass discs were not being similarly treated; at the School of Agriculture they were only being rinsed, not sterilized. A swab of the discs revealed an extremely high level of contamination, all of which appeared to be of the pseudomonad type. With the discs in use, the condensation falling back into the bowl served as an almost pure inoculum.

"Oxoid" Multodiscs showed that the organism was sensitive to tetracycline but not to penicillin, streptomycin or chloramphenicol. The glass discs were then scrubbed in hot water and given a final rinse in a tetracycline solution. There were no further cases of infection once this treatment had been initiated. The initial source, by which the discs became infected, could not be traced.

DISCUSSION

A search of the literature has failed to reveal a record of a *Pseudomonas* sp. initiating disease in *Linum usitatissimum*, although it has been recorded in the complex of organisms found in retting; the earliest reference being that of Hauman (3) who identified *B. fluorescens liquefaciens* (*P. fluorescens*) among the organisms. The present case occurred under highly unnatural conditions and there may have been several contributory factors, such as the high humidity and possible mechanical injury by the glass wool which led to an increased pathogenicity of the bacterium or susceptibility of the host.

The biochemical reactions which were tested do not permit identification of the species of pseudomonad, either by Dowson's (2) classification or from Bergey's Manual (1). The organism seems to resemble *P. fluorescens*, but the recent survey by Rhodes (4) of the properties of *P. fluorescens* shows that there is wide variation within the species. This, in turn, casts doubt on the validity of many of the species of *Pseudomonas*, which are named and mainly identified by their host. There has therefore been no attempt made to create a new species of pseudomonad here, or to identify it any further with an existing one.

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THE RENIFORM NEMATODE MAY BE A SERIOUS PEST OF THE SWEETPOTATO[1]

W. J. Martin

Results of recent greenhouse pot experiments have shown that the reniform nematode, Rotylenchulus reniformis Linford & Oliveira, 1940, may cause severe damage to sweetpotato, Ipomoea batatas. This appears to be the first report that sweetpotato is a host of the reniform nematode. The wooden rose, I. tuberosa, was reported as a host of the reniform nematode in Hawaii (2).

The reniform nematode has been found to occur in four cultivated fields in Louisiana, three in the Baton Rouge area (1) and one at Chennyville. The discovery of the reniform nematode in a field adjacent to experimental plots of sweetpotato in the Baton Rouge area prompted the investigation of the possible effects of this nematode on sweetpotato.

An experiment was conducted using 12 six-inch clay pots containing sterilized soil. The following three treatments were given to the soil in each of four pots: (a) non-infested controls, (b) infested with 2000 larvae of R. reniformis, and (c) infested with 8000 larvae of R. reniformis. On July 1, 1959 one cutting of the sweetpotato variety Unit 1 Porto Rico was planted in each of the 12 pots.

On August 27 a few roots were removed from one of the pots infested with 8000 nematodes. Although macroscopic examination of these roots failed to reveal evidence of infection, careful microscopic examination revealed the presence of a large number of nematodes in various stages of development on the root surface (Fig. 1).

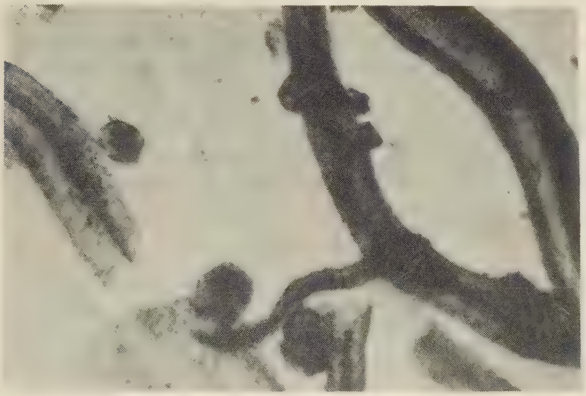

FIGURE 1. Mature females of Rotylenchulus reniformis on sweetpotato rootlets.

On December 2, 1959 roots were recovered from each of the pots. Roots from non-infested soil were abundant and exhibited little or no necrosis or discoloration, whereas roots from the infested soil were sparse, necrotic and discolored, with very few feeder roots. The mean weight in grams of roots from each treatment were: 22.5 from the non-infested soil, 10.1 from soil infested with 2000 larvae, and 3.8 from soil infested with 8000 larvae. Counts were made of the larvae from a pint soil sample taken from each of the two series of infested soil. From these counts estimates of the number of larvae per pot in the series originally infested with 2000 and 8000 larvae were 114,000 and 173,000, respectively.

The exceptionally severe pathogenicity exhibited by R. reniformis to sweetpotato in the experiment indicates that the reniform nematode may be a very serious pest of sweetpotato.

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[1] The nematodes were identified and the photograph was made by Wray Birchfield.

COMPARATIVE EFFICIENCIES OF THREE METHODS FOR EXTRACTING NEMATODES FROM ROOT AND SOIL SAMPLES[1]

Simón Malo[2]

Summary

The efficiencies of three elutriation methods for extracting nematodes from root and soil samples were determined. The Christie and Perry method was found to be the most rapid, whereas the Seinhorst elutriator yielded nematode suspensions with the least debris. The modified Oostenbrink apparatus described by Tarjan, Simanton and Russell extracted almost twice as many nematodes as the other two methods and was judged to be best adapted for processing numerous samples.

INTRODUCTION

Prior to the inception of a large scale nematode survey, various methods for extracting nematodes from soil and root samples were considered (1, 2, 3, 4, 6). Of these methods, three were regarded as sufficiently accurate to warrant further investigation. These were the Seinhorst elutriator method (4), the combination sieving-funnel technique proposed by Christie and Perry (3), and a method using the modified Oostenbrink elutriator proposed by Tarjan, Simanton and Russell (6). In the present study these three techniques were compared for maximum nematode extraction, speed of operation, and general utility for the proposed nematode survey.

PROCEDURE

Four tests were conducted using five replicated samples for the first, 15 for the second, and 10 for each of the last two tests. Nematode-infested material consisted of soil and feeder roots obtained at a depth of 6 to 12 inches from a grapefruit tree growing in Lakeland sandy loam soil containing 2 percent organic matter. Roots were separated from soil, cut into pieces approximately an inch long, and then uniformly mixed with the soil. This homogeneous mixture was divided into 1-pint samples. Prior to processing, roots were again separated from soil with the aid of a 10-mesh U. S. Standard sieve, combined with water, and comminuted for 10 seconds in a Waring Blendor. This root suspension was recombined with the soil prior to or during processing by each of the methods investigated. The total number of nematodes recovered in the first two tests was counted, whereas the total number in the two subsequent tests was estimated from aliquot samples of 10 ml. Each sample consisted of 100 ml of nematode suspension.

Seinhorst Method: Samples were processed in the Seinhorst elutriator in the following manner: a flow of water of 20 ml per minute was maintained in the apparatus, using No. 20 hypodermic needles. Soil was elutriated for 30 minutes, after which the collected suspension was poured three times through a U. S. Standard 325-mesh sieve. Residues remaining on this sieve were transferred to "parachute" nylon cloth filters (approximately 325 mesh) mounted in the top half of a 10-cm Petri dish. Nematodes that left the residues were then counted the following morning.

Modified Oostenbrink (F. C. E. S.) Method: The modified Oostenbrink elutriator (6) that was devised at the Florida Citrus Experiment Station is hereafter referred to as the F. C. E. S. method. Samples were processed in the manner previously reported (6), except for certain recently developed modifications that enhance the workability of the apparatus[3]. First, the soil sample deposited on the 10-mesh sieve was passed into the washing tube, using a standard household clothes spray nozzle connected to a water source. The entire sample was flushed into the tube as quickly as possible using the above hand-operated arrangement. This replaced the previous system which utilized a stationary sprayhead assembly of three whirljet nozzles so as to wash

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[1]Florida Agricultural Experiment Station Journal Series, No. 1061.

[2]Formerly Research Assistant, Citrus Experiment Station, now graduate student, Department of Fruit Crops, University of Florida, Gainesville.

[3]This constitutes the first report of these modifications developed by A. C. Tarjan, which supplements the original description of the apparatus (6).

down the soil slowly. Secondly, a 2-foot long, polished, clear plexiglass tube (with an inside diameter of 2 1/2 inches and a wall thickness of 1/8 inch) replaced the glass tube previously employed. The substitution eliminated a potential source of breakage. Finally, the lower nozzle assembly mounted at the base of the plexiglass tube was changed from the two opposing nozzle type, illustrated in the original publication, to a "T" sprayhead arrangement. This new arrangement utilizes a 1/8 G 3.5 nozzle directed upward and mounted on the middle upper surface of the sprayhead, and two 1/8 GG 3001.4 nozzles directed downward and mounted at the ends of the lower surface of the sprayhead. The upward nozzle is a standard Fulljet type with an orifice of 0.067 inch while the two lower nozzles are Fulljet Injector types with orifices of 0.026 inch (procurable from Spraying Systems Co., 3201 Randolph Street, Bellwood, Illinois). Samples were processed for 10 minutes, after which residues remaining on the sieves were collected, resuspended in water, and then passed three times through a 325-mesh sieve and placed on "parachute" nylon cloth filters mounted in Baermann funnels. The following morning liquid contents of funnels were passed through No. 41 Whatman filter paper placed in a Büchner funnel. Nematodes retained on the filter paper were counted after being washed into Syracuse watch glasses.

Christie and Perry Method: Samples were processed in the recommended manner (3) except that, as previously specified, the roots were comminuted in a Waring Blendor and then recombined with the soil. After water was added to the soil sample and the mixture thoroughly roiled, the heavier soil particles were allowed to settle for 10 seconds. The supernatant was then poured through a 60-mesh sieve, nested above a 325-mesh sieve. Residues remaining on the sieves were collected and further processed in Baermann funnels as previously described for the F.C.E.S. method.

DISCUSSION OF RESULTS

The first test, which utilized only five replicated soil samples per treatment, resulted in an average count per sample of 418 nematodes for the Christie and Perry method, 612 for the Seinhorst method, and 1117 for the F.C.E.S. method.

These results motivated the second test, which was similar in purpose but utilized 15 replicated samples per treatment. Due to the experimental design of these tests it was necessary to process and count the nematodes from samples in one experimental treatment before going on to the next treatment. Thus the Seinhorst method treatment was completed 2 days before the F.C.E.S. method and 6 days before the Christie and Perry method. Nematode counts from the Seinhorst method averaged 543, from the F.C.E.S. method 630, and from the Christie and Perry method, surprisingly 1025, in contrast to the results of the first test. On the theory that the disparity in counts may have been due to reproduction of nematodes in the original volume of soil used for this test, two additional samples were processed by both the F.C.E.S. method and the Seinhorst method immediately following acquisition of the results from the Christie and Perry treatment. The former method was now found to yield an average of 2465 nematodes, while the latter gave 1982 nematodes. It was apparent that storage of the soil and root mixture had resulted in a pronounced increase of the nematode population. These results confirm previous findings (5).

The third test was designed to minimize the delay in processing samples and thus eliminate increases of nematodes in the soil and root mixture prior to assay. Ten replicated samples were processed by each of the three methods within 48 hours. Results shown in Table 1 demonstrate the superiority of the F.C.E.S. method over the other two methods in regard to the total numbers of nematodes extracted from the soil and root samples. Here the average number of nematodes obtained by the F.C.E.S. method is significantly higher at the 1 percent level than the average numbers of nematodes obtained by the two other methods.

When the Christie and Perry method had been conducted previously, the soil samples had been washed only once and the supernatant passed through the sieves a single time. An attempt was made to improve the efficiency of this method by washing each soil sample three times and passing the supernatant liquid twice through the sieves. The remainder of the procedure was the same as that already described. Ten replicated soil and root samples were processed by this modified Christie and Perry method and 10 were processed by the F.C.E.S. method. An average of 179 nematodes per sample were obtained by the Christie and Perry method whereas an average of 290 nematodes were obtained from the F.C.E.S. method; this difference was significant at the 1 percent level.

Table 1. Comparison of the numbers of nematodes extracted from roots and soil by each of three elutriation methods in the third test.

Replicate	Seinhorst method	Christie and Perry method	F.C.E.S. method
1.	1275	800	1385
2.	520	890	1090
3.	800	300	1370
4.	750	660	1020
5.	690	570	1860
6.	350	600	1000
7.	390	930	1080
8.	530	780	1300
9.	550	490	980
10.	890	890	1130
MEAN	675	691	1222**

*L.S.D. at 5% = 227
**L.S.D. at 1% = 311

Most nematodes encountered were free living forms, however a few Dorylaims and a few plant parasitic nematodes were also found. *Tylenchulus semipenetrans* was not observed.

CONCLUSIONS

The normal nematode survey requires that a maximum number of samples be processed with a reasonable degree of efficiency, with a minimum amount of time and labor, using readily accessible equipment. The higher nematode counts obtained in these tests by the F.C.E.S. method, in contrast to the Seinhorst and Christie and Perry methods, proved its superiority, at least with regard to extracting larger numbers of nematodes from the samples used. In regard to the time required to process a single sample, the Seinhorst method, which required at least 30 minutes per sample, proved least desirable. The F.C.E.S. method required 12 minutes for a complete processing of one sample from start to deposition in the Baermann funnel, while the Christie and Perry method required about 8 minutes for the same operation. Since the F.C.E.S. and the Christie and Perry methods utilize relatively durable equipment, they are superior to the Seinhorst method, which uses specially prepared glass tubes that are easily broken in a moment of haste.

The F.C.E.S. technique appears to compare favorably with the other two methods in all of the considerations determined. Perhaps the primary consideration in favor of the F.C.E.S. method, however, is its greater efficiency in extracting nematodes; thus enabling the investigator to obtain more accurate knowledge about the kinds and numbers of nematodes within a given sample.

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PRUNUS INJUCUNDA SMALL AND ACER RUBRUM L., NATURAL HOSTS OF PEACH ROSETTE VIRUS

Glenn KenKnight

Prior to 1953 the only known wild host of peach rosette virus in Georgia was Prunus angustifolia Marsh. This wild plum is a sun-loving species that propagates by underground stolons as well as by seed and forms dense thickets that often occur near peach orchards. In brief surveys conducted in central Georgia in 1951-1959, peach rosette was found in low incidence in this plum in many localities.

In 1953 what later proved to be peach rosette was observed also in hog plum (Prunus injucunda Small) and a red maple (Acer rubrum) tree. Although in one locality in Crawford County several hundred P. injucunda died of peach rosette in 1953-59, the disease was not found in that host elsewhere. Only four rosetted Acer rubrum were found in 1953-59: two in Crawford County in association with rosetted P. injucunda, one in Peach County far from any peach or plum trees, and one in Houston County in association with rosetted P. angustifolia.

P. injucunda developed symptoms of peach rosette slowly and were affected several years before they died. Of 30 naturally affected trees tagged in 1953, 12 were alive in 1958 but all were dead in 1959. Symptoms consisted of severe stunting, which resulted in dense green foliage in spring. The foliage turned reddish in summer and yellowed before death.

Acer rubrum developed symptoms of peach rosette slowly except that only about 1 year was required for systemic expression to develop in seven small trees approach-grafted to rosetted peach, plum, or Acer rubrum trees. Short terminal growth of twigs of the trees so grafted resulted in the production of dense foliage closely resembling that of a naturally infected tree.

Peach rosette virus was transmitted from three diseased P. injucunda trees to peach and serially transmitted to apricot, P. angustifolia, P. hortulana Bailey, and Acer rubrum.

The only direct transmission of peach rosette virus from Acer rubrum to a species of Prunus was to Methley plum (P. salicina Lindl.). In 1954 five Methley plum trees were inoculated with buds from an affected Acer rubrum. This tree, located in Crawford County, was observed for several years before the other similarly affected trees were found. In 1957, after an incubation of 3 years, one Methley tree developed characteristic symptoms of peach rosette, and serial transmissions were made of the virus to Methley plum, peach, apricot, P. angustifolia, P. hortulana, and Acer rubrum.

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"FUSARIAL HEAD BLIGHT", SERIOUS DISEASE OF WHEAT IN GUATEMALA

Eugenio Schieber and Astolfo Fumagalli

Gibberella and Fusarium head blight appeared for the first time as a serious outbreak in central Guatemala. The scab developed at the end of the rainy season because of the high humidity and temperature in a wheat planted at an elevation of 4000 feet above sea level. Commonly wheat is grown in Guatemala at an altitude ranging from 6000 to 9000 feet; this explains in part the recent outbreak. The affected area (440 acres) was planted with spring wheat varieties Lerma Rojo and Guateian 2809. The first variety is an introduction from the Oficina de Estudios Especiales (The Rockefeller Foundation, Mexico), and the second one is a new improved selection (Supremo x Kenya) x (Perú x Supremo) x Perú II 2809. Losses amounting to 70 to 90 percent on both varieties were observed. These varieties did not show any attack by the disease at the normal altitudes where wheat is grown in Guatemala. In the field conidial development was abundant and the diseased spikelets showed the characteristic pink or salmon-pink cast[1]. Perithecia were also found in the field abundantly. At the time of harvesting the wheat kernels were shriveled and unsuitable for milling purposes. Isolations made in the laboratory proved to be Fusarium roseum f. cerealis (Cke.) Snyder & Hansen.

Because wheat is one of the important basic food crops in the country, the testing of new improved varieties at lower altitudes with higher temperature and humidity should be considered in order to find resistance to this disease[2].

INSTITUTO AGROPECUARIO NACIONAL, GUATEMALA, GUATEMALA C. A.

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THE OCCURRENCE OF ANGULAR LEAF-SPOT OF SESAME IN PANAMA

Juan B. Ferrer

A severe outbreak of angular leaf-spot of sesame was encountered for the first time in Panama during a disease survey conducted in early June of 1959. The disease completely burned the leaves, greatly reducing yields. Potentially this seems to be the most serious foliage disease affecting sesame under our climatic conditions.

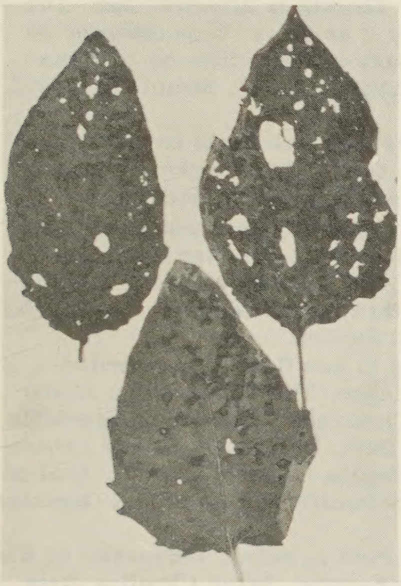

FIGURE 1. Sesame leaflets showing diseased areas infected with angular leaf-spot.

Angular leaf-spot of sesame has been recently described from India as caused by Cercospora sesamicola Mohanty, and as different from C. sesami Zim[1]. Leaf spots observed here were numerous, dull brown, without a distinct border, angular to irregular, vein-limited, small at first then coalescing until the entire leaf dies (Fig. 1). The fungus showed small brown stromata; fascicles compact and dense; pale brown conidiophores; conidia almost linear, long, subhyaline to very pale in color, slightly curved, $2\text{-}3\mu \times 20\text{-}200\mu$[2].

This disease was found to be seed-transmitted and most likely introduced into Panama on seeds from Nicaragua. The different varieties showed differences in susceptibility to this disease under field conditions. Use of tolerant varieties would probably be the best control. Further studies are underway to test variety reaction to this disease.

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[1] Mohanty, N. N. 1958. Cercospora leaf spot of sesame. (Abst.) Rev. Appl. Mycol. 38(9-10): 613.
[2] Acknowledgment is due to Dr. C. Chupp who kindly identified this fungus.

A WORLD SURVEY OF PHYTOPHTHORA PALMIVORA ON COCOA

P. D. Turner

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WAC

At the West African Cocoa Research Institute, Ghana, an extensive survey is in progress of possible variation in Phytophthora palmivora Butl., the causal fungus of black pod disease and other infections of cocoa. Experiments to date have been confined mainly to isolates from most of the cocoa-producing countries of West Africa, and marked differences have been found between the dominant types of pathogen in the region. Broadly speaking, the results have shown that cocoa in one group of countries, composed of Ghana, Ivory Coast and Sierra Leone, is attacked by a type of isolate very unlike that found on cocoa in another group, consisting of Nigeria, Southern and French Cameroons, Fernando Po and probably Gabon. The differences are morphological, physiological and pathological.

An extension of this work is planned to compare West African isolates with cultures from as many different countries as possible. Limited comparison has already been made with cultures and infected material received from other cocoa-growing areas. For example, isolates from Panama appear to be very similar to the Ghanaian type of isolate in production of oospores when grown with the Nigerian type and in all other characters tested. Preliminary results from these and other cultures have confirmed and amplified the findings on West African isolates by the range of oospore production in mixed cultures.

Stringent phytosanitary precautions are maintained; all tests involving foreign isolates are carried out in the laboratory under controlled conditions, and all material is sterilized by autoclaving before disposal. Samples of the fungus, either as diseased tissue or in pure culture, are welcomed from any source, including specimens from hosts other than Theobroma.

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BOOK REVIEW

"THE DISEASED PLANT." Volume I of a three-volume advanced treatise on Plant Pathology, Edited by J. G. Horsfall and A. E. Dimond. Published by Academic Press, New York-London. xiv + 674 pages. 1959. Price $22.

Leafing through this volume to such chapter headings as "Growth Is Affected" and "The Host is Starved", the seasoned pathologist might decide to lay it aside as "something for beginners." That is, until he notes that these simply-titled chapters are written by such distinguished scientists as Armin C. Braun of the Rockefeller Institute and C. Sempio of Italy's University of Perugia.

What the pathologist will find as he gets into this 600-plus page volume is an exhaustive and authoritative discourse on the diseased plant, written for the creative worker. He also may find it a somewhat revolutionary approach to the larger subject of plant pathology.

Here, in the first volume -- there are to be three -- is brought together pertinent knowledge from nearly a score of the world's leading biological scientists. Volumes II and III, scheduled for release early this year, are titled respectively, "The Pathogen" and "The Diseased Population -- Epidemics and Control." Editors of the three volumes are J. G. Horsfall and A. E. Dimond of the Connecticut Agricultural Experiment Station.

In addition to Dr. Sempio, foreign scientists contributing to this first volume include: J. G. ten Houten, Institute of Pathological Research, Wageningen, The Netherlands; Akhtar Husain, Regional Research Center, Kanpur, India; Antonio Ciccarone, Institute of Vegetable Pathology, Bari, Italy; D. Subramanian and L. Sarashwathi-Devi, University Botany Laboratory, Madras, India; Ikuzo Uritani and Takashi Akazawa of Nagoya University and S. Akai of Kyoto University, Japan; and K. O. Muller, Commonwealth Scientific and Industrial Organization, Canberra, Australia.

American contributors are: Dr. Braun; G. W. Keitt and Paul J. Allen, University of Wisconsin; K. Starr Chester, Alton Box Board Company; Arthur Kelman, North Carolina State College; C. E. Yarwood, University of California; F. L. Howard, University of Rhode Island, and the editors.

"The Diseased Plant" represents a major achievement for Editors Horsfall and Dimond. They have done a professional job of fitting together contributions from around the globe into a comprehensive and connected presentation. Moreover, they have determinedly tossed aside current concepts of the form that books on plant pathology should take in favor of what they believe to be a more logical organization that will help re-establish the semantic bases of the science.

Drs. Horsfall and Dimond disclose the theme of the first volume and of the entire treatise in the first chapter, where they explain why they have commenced with the diseased plant, instead of following the classical approach through the pathogen. Their sound reason: The diseased plant, not the pathogen, is the central theme of the subject of plant pathology. They define "disease" as a "process" and pick their chapter headings to conform to this idea -- "Tissue is Disintegrated", "Reproduction is Affected", for example.

Volume I covers the processes that result in diseased plants, and gives specific attention to such related areas as the histology, physiology, and biochemistry of defense in plants; hypersensitivity; predisposition; and therapy. The broader role of the diseased plant in world progress is dealt with in chapters on the scope and contribution and on the history of plant pathology.

"The Diseased Plant" is not a handbook. It is meant to be read from cover to cover. Each contribution is followed by a bibliography for those who would seek more information. The style of writing is the style of the individual contributors, with an assist from the editors, who have seen to it that all of the writing is up to a high standard. -- PAUL R. MILLER